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THE DIAGNOSIS AND MANAGEMENT OF ANGINA PECTORIS *

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It will be possible only to attempt to emphasize a few important practical points relating to the diagnosis and management of angina pectoris.

Some of the correctible sources of error in the diagnosis of this very common ailment are as follows:

1. Popular misconception regarding the symptomatology of the disease.
2. Difficulties in electrocardiographic interpretation.
3. Misconception regarding the action of nitroglycerin.

SYMPTOMATOLOGY

Too many persons, physicians, nurses, and laymen alike, apparently labor under the misapprehension that the pain of angina pectoris is always severe. Actually it is frequently not at all severe and because of this it is often dismissed as unimportant, or attributed to "gas", "indigestion", neuralgia or "tension". Too many persons apparently labor under the misapprehension that the pain in angina pectoris is located either in the anterior chest wall in the midline or over the heart, or in the left arm or both arms. Actually the pain may be located only in the jaw, the throat, behind either shoulder, in the back between the shoulder blades or at the base of the neck, or in any location or combination of locations anywhere in the body above the navel. More detailed description may be found elsewhere. The chief distinguishing characteristic of the symptomatology of angina pectoris is furnished by the fact that for each individual patient the discomfort always begins in the same area with each attack. In addition, if the discomfort radiates to other areas, then the manner in which it radiates is also the same in every attack, although the extent of the radiation depends upon the duration and the severity of the attack.

The character of the discomfort may be described as sharp pain, a sensation of pressure or a lump, burning, numbness, or tingling. It may last only a few seconds or an hour or more. It may be intermittent, consisting of mild discomfort lasting a few minutes and recurring at intervals of a few minutes.

There is much popular misconception regarding the quantitative relation of the attacks to exertion. Almost everyone knows that the attacks are precipitated by emotional tension and by exertion. When the attacks are precipitated by ex-

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ertion many have assumed that the likelihood of precipitation of an attack by activity is proportional to the quantity of exertion involved in that activity. Although this is frequently true, the relation between the attacks and the activity which precipitates them is often more specific than quantitative. Many persons have pain as a result of walking one block who can climb flights of stairs without having the slightest discomfort. Carpenters and automobile mechanics who have had no attacks while working hard have reported that they have them regularly while walking to the bus stop, or while maneuvering the car into a parking space. These inconsistencies are so common that they may almost be regarded as characteristic. Yet errors are commonly made because of the mistaken expectation of consistency in the relation between the attacks and the exertion which precipitates them.

THE ACTION OF NITROGLYCERIN

When a patient suffers paroxysmal attacks of discomfort in the upper half of the body in each of which the location and manner of radiation of the discomfort (if any) is always the same, and if these attacks are invariably relieved within two to four minutes by nitroglycerin, then that patient suffers with angina pectoris. Necessarily, the conclusion that nitroglycerin has relieved the attacks in dramatic fashion can only be arrived at if it is known that previous attacks have lasted much longer than the time required for nitroglycerin to act. In some cases anginal attacks last less than five or six minutes. Obviously, in these the effect of nitroglycerin cannot be used as diagnostic evidence. We must resort to the electrocardiogram.

Some have nursed the notion that nitroglycerin relieves the pain of biliary colic, intestinal colic, and other conditions. I can only say that I have not seen nitroglycerin relieve any paroxysm of pain in dramatic fashion except in the presence of angina pectoris.

ELECTROCARDIOGRAPHIC INTERPRETATION

It is probably safe to say that no other one thing in the field of medicine has given rise to more errors than misinterpretation of the electrocardiogram. Our errors occur among that great number of patients whose discomfort is either not in the classic location or cannot be proved to respond to nitroglycerin. When confronted by this problem it is necessary to know: (1) that the electrocardiogram is most frequently normal between attacks and that the diagnosis is evident *only if an electrocardiogram is made during an attack;* and (2) that if the electrocardiogram is abnormal between attacks the presenting symptom may still be due to noncardiac disease unless serial interim tracings show significant progressive or regressive changes.

Failure to realize that in many cases the electrocardiogram shows changes only during the attacks has led to many unfortunate errors. One patient with epigastric pain radiating to the back was studied over a period of three months including three admissions to two hospitals, numerous electrocardiograms, two gall bladder visualizations, two gastrointestinal series, two barium enemas, and an imposing array of other laboratory procedures. It was finally suggested that he have an exploratory laparotomy. The most important fact discovered in taking his history was that none of the numerous electrocardiograms had been made during an attack of pain. Accordingly arrangements were made to record an electrocardiogram during his next attack. This occurred some thirty-six hours after he was first seen. The electrocardiograms made at this time are seen in Figure 1. There can now be no doubt about the diagnosis. Needless to say he did not have a laparotomy.

If it is impossible to make an electrocardiogram during a spontaneous attack attempts are made to induce an attack by reproducing in the office the conditions under which the attacks occur spontaneously. These conditions may be specific

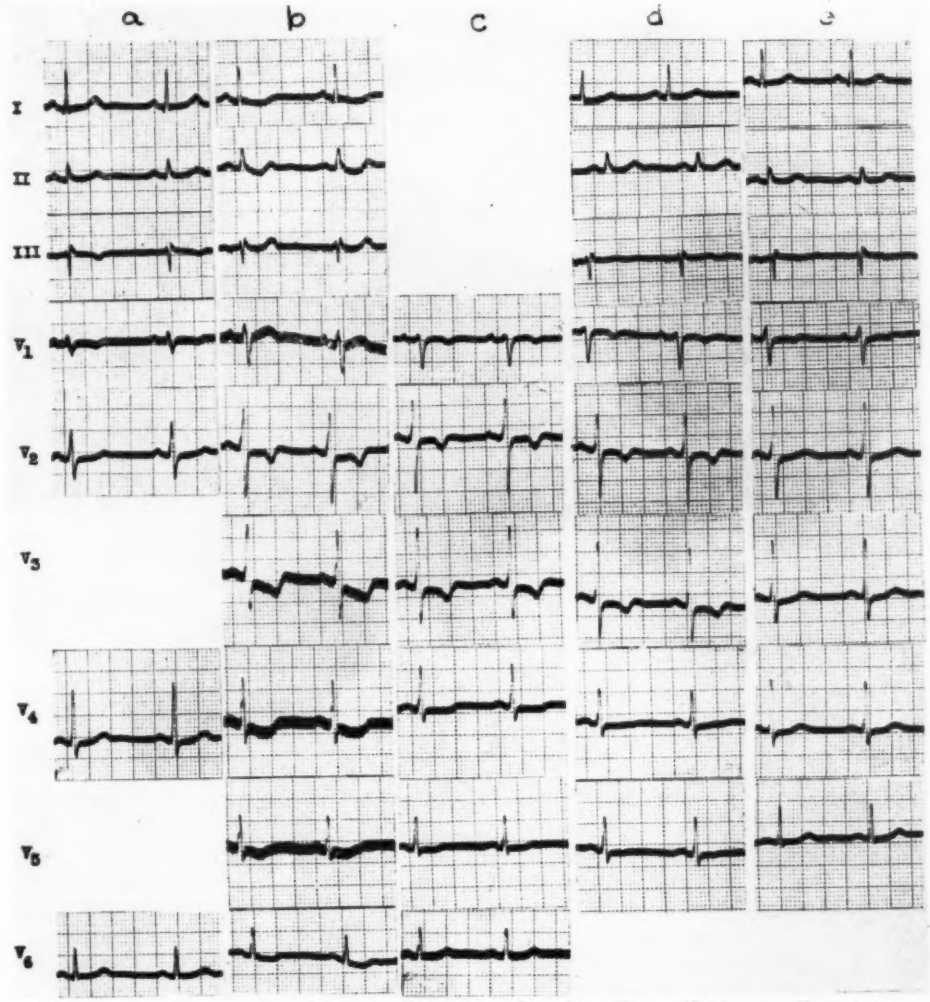

Figure 1—**a** The routine electrocardiogram made when the patient was first seen. **b, c, d,** and **e** were made in sequence over a period of 20 minutes during and after a spontaneous attack of angina.

Exercise tracings made 20 hours before were negative.

exertions, the taking of food, exposure to cold and combinations of these. Even when no relation to exertion is obvious in the history, electrocardiograms are made before, immediately after, and five and ten minutes after twenty deep knee bends done very rapidly. This constitutes greater exertion than that involved in the performance of the Master's double two step test. If pain occurs at any time the exercise is stopped and an electrocardiogram is recorded. Occasionally the diagnosis is revealed in this way. However, it is extremely important to point out that many patients whose electrocardiograms remained normal following twenty deep knee bends, within a few days, showed, during a spontaneous attack, electrocardiographic changes which were pathognomonic of angina pectoris (Figure 1). Evaluation of exercise electrocardiograms requires detailed knowledge of the effects of exercise on the electrocardiogram in normal persons. Unfortunately,

normal changes between the control and exercise tracing are frequently attributed to coronary disease.

If no opportunity is had to make observations during a spontaneous attack and no attack can be precipitated by exercise or other methods the electrocardiogram may be of little help. The interim tracing may be normal or abnormal. Even in the latter case the diagnosis is not assured because many persons of middle age or older have arteriosclerotic electrocardiographic changes which are not necessarily related to the symptoms. If over a period of several days progressive or regressive changes in the electrocardiogram are observed the diagnosis may be made on this basis. However, in order to be able to evaluate these serial changes it is necessary to be able to distinguish changes due to a variety of nonpathological factors such as rate change, food, exertion, smoking and changes in cardiac position from those due to disease. Indeed, familiarity with the effects of these factors is necessary to the interpretation of the single electrocardiogram.

Occasionally the electrocardiogram shows no change even at the height of a very severe attack. Only repeated dramatic relief by nitroglycerin or positive electrocardiographic signs in a subsequent attack can then make the diagnosis.

Some of the most difficult problems are presented by those patients who are known to have heart disease (even previous myocardial infarctions) and who develop pains in the chest, epigastrium or other areas which may actually be due to noncardiac disease. Here, again electrocardiographic observation during the attacks may be of the utmost importance. If repeated observation shows that little or no change in the electrocardiogram occurs other causes of the pain should be sought. Space does not permit full discussion of this problem.

THE MANAGEMENT OF ANGINA PECTORIS

The object of management of this condition is the preservation of the normal functional state of the individual. The most important problem is encountered as soon as the diagnosis is made, and is constituted by the patient's emotional reaction to the information that he has angina pectoris. His apprehensions, generally expressed in terms of what he has heard about other cases in his family or among his friends, or in terms of what he has heard from others or has read in lay journals, must be explored and exposed in detail. Intelligent reassurance must be offered and if the patient is in a nonlaborious occupation he must be taught from the very beginning that he will be able to continue with his usual occupation.

Nitroglycerin is used to abort the attacks and the patient must be taught that it must be used as early in the attack as possible. This advice usually has to be repeated several times because many patients attempt to "get by" attacks without the use of nitroglycerin in the mistaken notion that their progress can be measured by how few nitroglycerin tablets they use. Experience has taught that this method of keeping score is self-defeating, for if the patient uses nitroglycerin at the very inception of every attack it is generally found that the attacks will soon occur less and less frequently. The patient must be taught to treat the attacks casually, to abort the pain as early as possible in the attack and to forget that the attack has occurred as soon as the discomfort is gone. The development of such an attitude usually requires several months and the degree of success which can be realized in attaining it depends upon the personality of the patient and upon the degree to which the physician encourages it. Such patients should not be treated by physicians who are fearful that their reputations will be damaged if a patient whom they have encouraged to activity happens to drop dead during that activity. Such physicians create, unnecessarily, too many psychologically determined cardiac cripples. This is not to say that judgment should not be exercised. If a patient is having *very* frequent attacks of severe

onset it is wise to advise bed rest or home rest for a trial period but even in these cases the patient is advised that he is expected to return to work within a relatively short period of time. Most often the frequency of the attacks is reduced to a marked extent within a few days or a week perhaps with the help of nitroglyn and sedation. If after a week has passed the attacks occur once or twice daily and there is no significant change in the electrocardiogram the patient is returned to nonlaborious work on a limited basis. The limit is raised as rapidly as possible.

If the attacks do not tend to subside rapidly or if they become more frequent during bed rest one must conclude that he is probably dealing with imminent infarction. Such patients should be treated with anticoagulants, so-called "coronary dilators", and sedation. Frequent electrocardiographic observations must be made to detect infarction if it occurs. This may take place slowly. In some cases infarction does not occur for a number of weeks if at all, and the morale of the patient and of the family becomes an enormous problem. Consultation with other reputable physicians in the neighborhood will be of some help in this regard. However, nothing can completely relieve the anxiety associated with the knowledge that one is sitting on a barrel of nitroglycerin. In a few of these severe cases very much prolonged duration of a period of very frequent attacks justifies the use of antithyroid measures such as tapazole, or radioactive iodine.

Fortunately, most cases fall in the first group described; the attacks subside in frequency within a relatively short period and the patient returns to function in the manner already related. Something should be said with regard to permanent restrictions. This is a large subject and can only be touched upon here. It is important to encourage the patient to live as normally as possible. Nowhere else in medicine is it so clear that "a light heart lives longer". Never should a patient be handed a printed list of "don'ts". One should emphasize the things that he *can* do. Relief of tension is the greatest single factor in improvement. To this end family and business problems are discussed. To this end normal sexual relations are encouraged and three or four highballs daily are advised.

The most ridiculous advice that can be given a human being is "don't worry and don't get excited". These things are not under the control of the will and one cannot live again in the womb though many have tried. Circumstances give rise to worry and excitement regardless of the physician's advice. The effect of the advice is to tend to make the patient suppress his feelings instead of expressing them and suppressed feelings generate tension which is destructive. Another effect of such advice is that it frightens the patient; he feels that he must be fragile indeed if he cannot stand a bit of excitement—sudden death must be hovering over him.

The use of tobacco is a moot point in these cases. I do not prohibit the use of tobacco unless it causes attacks or unless it causes excessive coughing. Deprivation of smoking frequently causes heightened tension which is more threatening than the theoretically objectional effects of smoking tobacco.

I do not encourage physical exercises. Patients who have angina may be permitted much necessary activity but I see no sense in encouraging unnecessary physical exertion. I know of no well-founded scientific investigation that offers any basis for the notion that a middle-aged human being really needs to idly flex his muscles for any other than puerile psychological reasons.

Finally, it is well to stress the fact that the most destructive force in the life of many cardiac patients is the spouse who is constantly "on his neck" with don'ts. After all, there is no reason why he cannot open an ordinary window which is not stuck, move a chair which does not weigh twenty-five pounds, bend down to pick up something off the floor, or per-

form many other simple acts. He may even climb stairs if he can do so without discomfort.

A summary and conclusions for such a paper as this is unnecessary and would constitute a waste of space.

DISCUSSION

Dr. C. T. Yancey (Monroe): I wish to congratulate Dr Gardberg on the scope of this paper since he has emphasized many of the misconceptions relating to the diagnosis and management of a frequently found syndrome. Misdiagnosis of angina has many ramifications, and as alluded to by Dr. Gardberg, influences the psyche of an entire household when such a diagnosis is made. The economy and future planning for an entire family must be considered. Too much time cannot be spent in explaining what the condition is and how it should be managed from the standpoint of exertion, emotional problems, and the use of medications.

Frequent revaluation of the symptoms should be done since many other disease states too numerous to enumerate may simulate anginal syndrome. Careful history relating to the pulmonary, gastrointestinal tract and genitourinary tract may divulge a lead which will eventually disprove the anginal diagnosis and bring to light another disease state which is amenable to medical or surgical therapy. The coexistence of proven angina and another disease state poses a hazardous problem which should be evaluated carefully by one or more consultations before definite action is taken.

I thoroughly agree with the author that the use of the electrocardiogram has been a definite boon to the diagnosis of angina, but misinterpretations and miscorrelation of symptoms with the electrocardiogram have led to an erroneous diagnosis many times. The author's advice with regard to frequent use of the electrocardiogram especially during bouts of pain, is sound and scientific and should be employed more frequently. There are several methods by which electrocardiographic changes signifying coronary ischemia can be reproduced, but one should be cognizant that both false positive and false negative results can be recorded. Relief of induced or spontaneous pain by nitroglycerin is by far the most reliable method by which the diagnosis can be made.

The management of the patient during the early stages, after the diagnosis has been made, with reassurance, weight reduction if indicated, control of exertion and emotion, and the use of nitroglycerin have been emphasized by Dr. Gardberg but cannot be stressed too frequently. Also the treatment of precipitating causative factors especially those relating to the gastrointestinal tract and lungs should not be neglected. Diabetes mellitus coexistent with coronary ischemia deserves meticulous attention. Either overzealous use of insulin with resultant hypoglycemic reactions or neglect of hyperglycemia and acid base imbalance will aggravate the anginal syndrome and may lead to myocardial infarction.

One of the most interesting phenomenon in the history of the anginal syndrome is the reduction or relief of pain following an acute myocardial infarction. The area involved by infarction becomes necrotic and avascular therefore does not respond to ischemic changes as it did prior to the attack of infarction.

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THE DIAGNOSIS OF SICKLE CELL DISEASE IN PEDIATRICS

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INTRODUCTION

The significance of various percentages of erythrocyte sickling on the standard sickle cell preparation frequently raises unanswered questions on the pediatric service. It was therefore decided to review the charts of all pediatric patients who had electrophoretic determination of hemoglobin type here from January, 1955, when our laboratory began doing electrophoretic studies on hemoglobin, through June, 1957. Other questions which we hoped to answer from our own cases were: (1) What is the average hemoglobin level of patients with sickle cell anemia? (2) How does this compare with the hemoglobin level in sickle cell trait and with the "normal" hemoglobin of the same age, race, and area? (3) What other information helps to establish the diagnosis of sicklemia?

Up to this point, I have purposely used four different terms related to the entity which we are discussing. The wide use of all of these terms results in misunderstanding unless we define them and indicate the way in which each is used. "Sickle cell disease", sickle cell anemia", and "sicklemia" are used synonymously here to denote patients having sufficiently high percentage of hemoglobin S to produce symptoms from that cause alone. "Sickle cell trait" is used to denote patients with some hemoglobin S, but of insufficient quantity to produce symptoms. These would be clinical definitions. Defined in terms of genetics, a patient with sickle cell anemia is one who has received the gene for hemoglobin S from both parents, and a patient with sickle cell trait is one who has received the gene for hemoglobin S from only one parent. Usually the other gene is for hemoglobin A, the normal adult hemoglobin. However, it may be some other type such as, for example, hemoglobin C.[1] Combinations of the rarer types are beyond the scope of this paper.

A total of 111 charts were studied. These represented all pediatric patients who had had hemoglobin electrophoretic studies done through June, 1957, and twenty Negro pediatric patients chosen at random as a control. In order to analyze the data, the charts were divided as follows: (1) patients with 100 per cent hemoglobin S as determined by electrophoresis, (2) patients with combinations of hemoglobin S and hemoglobin A, (3) patients with 100 per cent A, and (4) the 20 charts used as a control. Table 1 shows the hospital diagnosis which had been made in each case. Three charts were eliminated because of conflicting results of electrophoresis when two or more determinations had been made.

TABLE 1
HOSPITAL DIAGNOSIS AND HEMOGLOBIN TYPE OF ALL PATIENTS STUDIED

	Sickle Cell Disease	Sickle Cell Trait	Other	Total
100% Hemoglobin A	1	0	27	28
S and A Hemoglobin	12	3	18	33
100% Hemoglobin S	25	0	2	27
Control	0	0	20	20
Eliminated	0	0	3	3
Total	38	3	70	111

FINDINGS

In examining the charts, the highest and lowest hemoglobin recorded for each patient was noted. This gives us two sets of figures to compare and was done in lieu of calculating the average hemoglobin of each patient. From these figures, we

TABLE 2
AVERAGE HEMOGLOBIN * ACCORDING TO HEMOGLOBIN TYPE

	High (Range)	Low (Range)
Control	12.1 (9.8-15.1)	9.7 (4.3-12.4)
100% A	10.5 (6.7-14.9)	7.6 (2.9-13.2)
S and A	11.4 (4.2-14.1)	8.5 (4.2-12.6)
100% S	10.1 (7.6-14.1)	5.0 (2.5-10.9)

* Determinations recorded in the birth admission are not included.

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have determined two average hemoglobins for each group—the average of the high hemoglobins and the average of the low hemoglobins. These figures are seen in Table 2 which also shows the ranges of hemoglobin level in each group. In the control group, the hemoglobin ranged from 4.3 grams per 100 cc. of blood to 15.1 grams per 100 cc. of blood. The average of the highest hemoglobins was 12.1 and the average of the lowest hemoglobins was 9.7. However, these should not be considered normal hemoglobin values since some of them are obviously much below normal and they are from hospital patients instead of well children. They are, simply, averages of a random hospital population. For those patients with 100 per cent hemoglobin S, the range of hemoglobin levels was not greatly different from the control. It was 2.5 to 14.1. However, the average high hemoglobin was 2.0 grams less than the control and the average low hemoglobin was 4.7 grams less than the control. It should be mentioned at this point that the 100 per cent S group received a total of 136 blood transfusions, which is an average of five transfusions per patient. Seventeen of the 27 in that group had blood within one month prior to the time that the highest hemoglobin was recorded. The group with 100 per cent A and those with combinations of S and A fall between the control group and those with 100 per cent S. Surprisingly, the 100 per cent A group was found to have lower values than the SA group. The hemoglobin levels in the SA group approach the control levels. A plausible explanation for this finding is that in the 100 per cent A group, the electrophoretic determination of hemoglobin type was often done in working up an anemic patient. In the SA group, it was more often done because of the finding of a positive sickle cell preparation which is often done as a routine procedure on Negro patients. In this entire group, only two blood transfusions were given. This finding leads to the impression that patients with less than 100 per cent hemoglobin S are not likely to have a marked, persistent anemia.

Table 3 gives a breakdown of the group having both hemoglobin S and hemoglobin A with the average hemoglobin for each percentage of hemoglobin S as well as the percentage at which sickling is first seen on a sickle cell preparation. Though this group is too small to establish any definite conclusions, it is readily seen that the groups with higher percentages of hemoglobin S do not have lower hemoglobins than those with a predominance of hemoglobin A. It is of interest, also, that of the 33 patients in this group, all except 1 had 60 per cent or less hemoglobin S. In other words, only 1 patient had between 60 per cent and 100 per cent. Only 3 had more than 50 per cent hemoglobin S. Most of the group, 27 of 33, had between 20 per cent and 50 per cent hemoglobin S. In reporting various percentages of the different types of hemoglobin, the technician must make an estimate unless it is all of one type. Therefore, these are not exact determinations. Because of the finding that most of this group have in the neighborhood of 50 per cent hemoglobin S or less and the relatively high average hemoglobin in this group, we can consider these patients to have sickle cell trait. This is substantiated by the paucity of symptoms and signs in this group as will be shown later.

TABLE 3
HEMOGLOBIN LEVEL ACCORDING TO AMOUNT OF HEMOGLOBIN S

Percentage of Hemoglobin S*	Hemoglobin High	Hemoglobin Low	Number in Each Group	Sickle Cell Preparation (Highest percentage recorded)
5%	11.3	9.8	1	Negative
11%	4.2	4.2	1	Not done
10-15%	9.9	9.0	1	Negative
20%	11.0	6.8	4	100%
30%	12.0	9.6	5	100%
40%	11.8	8.5	12	100%
50%	11.3	8.9	6	100%
51%	11.5	9.3	1	100%
60%	14.1	11.0	1	100%
60-80%	11.5	6.3	1	100%

* Hemoglobin A made up the remainder in all cases.

Going now to a consideration of the

significance of sickle cell preparations, we see the data in Table 4. Here, we find that 45 per cent of the preparations of the patients with sickle cell trait had 100 per cent sickling. Table 3 shows that this can occur with as little as 20 per cent hemoglobin S. Also, in Table 4 again, 11 per cent of those with sickle cell anemia had 50 per cent sickling or less, one as low as 5 per cent. These findings indicate that the percentage of sickling on a sickle cell preparation is not significant. A positive preparation of any degree is evidence that the patient has either sickle cell trait or sickle cell disease. Although not shown in the tables, it should be mentioned that of the 16 patients with 100 per cent hemoglobin A who had sickle cell preparations, sickling was reported in 2, 50 per cent in one instance and 100 per cent in the other. This shows that false positives may occasionally be reported, possibly due to error in the laboratory or in recording. Also, it is seen in Table 4 that 12 per cent of patients with sickle cell trait had negative sickle cell preparations.

Included also in Table 4 is a finding of greater significance than the sickle cell preparation—that of sickling on a routine Wright's stain blood smear. This was found only once among those with sickle cell trait, but was found 88 times among those with sickle cell disease. Twenty-one individuals (78 per cent) had this finding at least once. This is, by definition, sicklemia. Its absence does not rule out sickle cell disease, but finding it is strongly indicative that the patient has 100 per cent hemoglobin S.

Other laboratory data studied are red cell indices, red cell fragility, and serum bilirubin. These results are only for the patients with 100 per cent hemoglobin S. Bilirubin determination was done a total of 26 times, for 17 patients. Nineteen times (73 per cent) the total bilirubin was greater than one milligram per cent and 7 times (27 per cent) it was less than one milligram per cent. This excludes patients known to have hepatitis. Of 4 red cell fragility determinations, 3 had beginning hemolysis in a concentration of saline lower than the control and all had complete hemolysis in a concentration lower than the control. The red cell indices of 10 patients had been calculated. The averages are as follows: mean corpuscular volume, 77.5 cubic microns; mean corpuscular hemoglobin, 25.9 micromicrograms; and mean corpuscular hemoglobin concen-

TABLE 4
RESULTS OF SICKLE CELL PREPARATIONS AND INCIDENCE OF SICKLING ON WRIGHT'S STAIN BLOOD SMEAR ACCORDING TO HEMOGLOBIN TYPE

All Sickle Cell Preparations	Sickle Cell Trait (Hemoglobin S and A)		Sickle Cell Anemia (100% Hemoglobin S)	
	Number	Percentage	Number	Percentage
Negative	5	12%	0	0
1-50% Sickling	5	12%	6 (Lowest 5%)	11%
51-99% Sickling	13	31%	17	30%
100% Sickling	19	45%	33	59%
Total	42		56	
Results of Sickle Cell Preparation by Patient				
Negative	4	12%	0	0
Positive	26	79%	25	93%
Positive and Negative	1	3%	0	0
Not Done	2	6%	2	7%
Total	33		27	
Wright's Stain Blood Smear				
Total Positive	1		88	
Positive Individuals	1	3%	21	78%

tration, 34.6 per cent. The former two figures are slightly lower than normal and the latter figure is within normal range. This is the same type of abnormality which is found in iron deficiency anemia and is, therefore, of no diagnostic aid here.

Table 5 shows the signs and symptoms encountered in the patients having 100 per cent hemoglobin S as compared with those having both S and A. The symptoms which occurred most commonly in the sickle cell disease group were abdominal pain, pain in the arms or legs, joint pain, back pain, swelling in the arms or legs, and exertional dyspnea. The common physical findings were icterus, hepatomegaly, systolic murmur, splenomegaly, and chronic leg ulcer. Some of the same signs and symptoms occurred in the sickle cell trait group, but in a much smaller percentage of cases. More commonly, the findings in this group were referable to some unrelated disease and the finding of sickle cell trait was incidental. The signs and symptoms related to these other diseases are not shown in the table. Common findings in sickle cell anemia which are not listed in Table 5 were fever and elevated white blood cell count. It is felt that these were usually due to intercurrent infection which is common in sicklemia patients, probably because of a low resistance which, in turn, is due to inadequate hemoglobin. Pallor is also not listed and was, of course, a common finding.

SUMMARY AND CONCLUSIONS

On the basis of the information given above the following conclusions have been reached.

1. The average hemoglobin of hospitalized Negro pediatric patients in this area is from 9.7 to 12.1 grams per 100 cc. of blood.

2. Patients with a combination of S and A hemoglobin have, as a group, a hemoglobin level slightly lower than this.

3. Patients with 100 per cent hemoglobin S have an average hemoglobin of 5.0 to 10.1 grams. These figures would be much lower were it not for the fact that the patients had received an average of five transfusions each.

4. Patients with as little as 20 per cent

TABLE 5
SIGNS AND SYMPTOMS ACCORDING TO HEMOGLOBIN TYPE

Symptoms	100% Hemoglobin S		Hemoglobin S and A	
	Number	Percentage	Number	Percentage
Abdominal Pain	12	44%	3	9%
Limb Pain	11	41%	3	9%
Joint Pain	7	26%	3	9%
Back Pain	7	26%	1	3%
Limb Swelling	4	15%	1	3%
Exertional Dyspnea	4	15%	1	3%
Malaise	3	11%	1	3%
Anorexia	3	11%	0	0
Chest Pain	2	7%	2	6%
Epistaxis	2	7%	1	3%
Headache	2	7%	1	3%
Joint Swelling	1	4%	0	0
Face Swelling	1	4%	1	3%
Poor Development	1	4%	0	0
Abdominal Distention	1	4%	0	0
Signs				
Icterus	13	48%	0	0
Enlarged Liver	13	48%	1	3%
Systolic Murmur	12	44%	0	0
Enlarged Spleen	11	41%	2	6%
Leg Ulcers	4	15%	1	3%
Limb Tenderness	3	11%	0	0
Joint Tenderness	3	11%	0	0
Abdominal Tenderness	1	4%	0	0

hemoglobin S can have 100 per cent sickling on a sickle cell preparation.

5. Patients with 100 per cent hemoglobin S can have as little as 5 per cent sickling on a sickle cell preparation.

6. A positive sickle cell preparation usually indicates that the patient has either sickle cell trait or sickle cell disease. However, false positives do occur.

7. A negative sickle cell preparation probably rules out sickle cell anemia, but it does not rule out sickle cell trait.

8. The finding of sickled cells on a Wright's stain blood smear is practically diagnostic of sickle cell anemia, but the absence of this finding does not rule out the diagnosis.

9. In sicklemia, the total serum bilirubin is frequently elevated.

10. Based on only four cases, it appears that the red cells of patients with sickle cell disease are less fragile in hypotonic saline than those of normal controls.

11. Red blood cell indices do not appear to be of any diagnostic value in sickle cell disease. This is based on only 10 cases.

12. The common symptoms and signs of sickle cell disease are abdominal pain, limb pain, joint pain, back pain, limb swelling, exertional dyspnea, icterus, hepatomegaly, systolic murmur, splenomegaly, leg ulcer.

13. Patients with less than 100 per cent hemoglobin S usually have about one-half or less hemoglobin S, relatively high hemoglobin, relatively few symptoms, and seldom require a blood transfusion. For these reasons, we consider this group to have sickle cell trait.

Note: Acknowledgement is made to Dr. C. H. Webb and Dr. M. A. Bodron, Jr. for their valuable suggestions, and to Mrs. Aline Riley for her help in obtaining the charts.

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DIAGNOSIS OF ADRENAL TUMORS

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In recent years the physiology of the adrenal glands has been the subject of considerable study. As a result, interest has been renewed in the role of the adrenals in surgery and improvements have been made in the diagnosis and treatment of adrenal tumors. It is the diagnostic aspects of this problem with which this paper is concerned.

In order to approach the diagnosis of adrenal tumors in an intelligent manner, one must be aware of the types of adrenal tumors that can occur. These are listed in the classification of adrenal tumors in Table 1.

TABLE 1
CLASSIFICATION OF ADRENAL TUMORS

I.	Tumors of the Cortex
	A. Hyperfunctioning tumors
	B. Nonfunctioning tumors
II.	Tumors of the Medulla
	A. Functioning tumors
	1. Pheochromocytoma
	B. Nonfunctioning tumors
	1. Neuroblastoma
	2. Ganglioneuroma

TUMORS OF THE ADRENAL CORTEX

To make a diagnosis of adrenal tumor, one must be aware of the varied clinical manifestations of the different types of tumors. *The nonfuctioning cortical tumors* give signs and symptoms of tumor only, such as mass, pain, pressure symptoms, or metastases. These tumors are usually much larger than the functioning tumors at the time of diagnosis. This is due to the fact that there are no endocrine symptoms to call early attention to the possibility of a tumor.

The hyperfunctioning tumors of the adrenal cortex have frequently been discussed in the past with reference to "the adrenal cortical syndrome." It is now ap-

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parent that there is no single syndrome which is characteristic of adrenal cortical hyperfunction. The symptoms which may accompany tumors of the adrenal cortex have been divided by Kepler and his associates [5] into those dependent on age and sex and those which are independent of age and sex. The former category includes homologous sexual and somatic precocity in boys, sexual and somatic precocity in girls (usually heterologous), feminization of men and various degrees of virilism in women. The signs and symptoms, independent of age and sex, include altered habitus (facial and abdominal obesity, kyphosis, and thin arms and legs), purplish striae, hypertension, osteoporosis, diabetes, acne, polycythemia, ecchymosis, fatigue, and muscular atrophy.

Any combination of these symptoms can occur so that the clinical pictures with hyperfunction of the adrenal cortex may be extremely varied. Kenyon [4] has noted the following types of clinical syndrome. (1) The adrenogenital syndrome. This term may properly be applied in cases in which changes in the direction of increased masculinity dominate the clinical picture. (2) Cushing's syndrome. This refers to the clinical picture described by Cushing and not specifically to the pathologic basis of the syndrome. In the past it was thought that Cushing's syndrome was always associated with a basophilic tumor of the anterior lobe of the pituitary gland. Experience has shown that this syndrome may occur in the absence of pituitary tumor but is probably always associated with hyperfunction of the adrenal cortex. (3) Mixed clinical pictures, including features of the first two. (4) Type characterized by a single or isolated endocrine manifestation (diabetes or amenorrhea). (5) Feminizing syndromes—feminization of men or homologous sexual precocity in girls.

There are a number of cases in which symptoms will not fit into any of the above categories because the clinical picture is incomplete or confused and these will be difficult to classify accurately.

TUMORS OF THE ADRENAL MEDULLA

Pheochromocytoma, chromaffinoma, and paraganglioma are the names which have been used to designate the *functioning tumor of the adrenal medulla.* These tumors produce their clinical picture by the outpouring of an epinephrine substance which causes hypertension. Consequently the symptoms of these patients are primarily those of hypertension. In the classical picture, the hypertension is paroxysmal, may come on suddenly associated with striking, severe symptoms and the patient may be entirely well between attacks. On the other hand, in one-third of the cases the hypertension is usually sustained and the patient will have the symptoms of essential hypertension. Among these are headache, palpitation, vomiting, sweating, dyspnea, weakness, pallor, dizziness, substernal pain, failing vision and so forth. In those patients with paroxysmal symptoms the interval between attacks may vary from minutes to months. The attacks may be initiated spontaneously or may be precipitated by external pressure or by assumption of a particular position. Objective findings during an attack may include anxiety, pale, cool skin, dilated pupils, rapid pulse, elevated blood pressure and distended neck veins. The blood pressure elevation may be systolic, diastolic or both. The eye grounds vary from normal to group IV. The heart may or may not be enlarged. The basal metabolic rate is usually elevated and a fasting blood sugar of above 120 is not uncommon during the attacks.

Pheochromocytomas are not common, but it is estimated that they are responsible for one-half to 1 per cent of all deaths due to hypertension each year.[3] They may be found at any age and are reported from 5 months to 72 years, but their greatest incidence is during the fifth decade.[3] There is some indication of a familial tendency with 3 bilateral cases reported in siblings.[3] There has also seemed to be some relationship to gallbladder disease because in 1 series of 50 patients with pheochromocytoma, 7 had pathologic gallbladders.[6] Pheochromocytomas most commonly occur in

the adrenal medulla but may occur anywhere chromaffin tissue is found. They have been called the 10 per cent tumors—approximately 10 per cent are bilateral, 10 per cent are located in extra-adrenal areas and 10 per cent are malignant.

One must remember these tumors to make the diagnosis. Suspicion should be aroused in paroxysmal hypertension, sustained hypertension of short duration (especially with superimposed paroxysms, excessive sweating, palpitation or tachycardia, episodes of hyperthermia, hypermetabolism, or alteration of the carbohydrate metabolism), hypertension in children, excessive systemic response to histamine when given for gastric analysis and unusual blood pressure response as during anesthetic induction. More specific diagnostic aids will be discussed below.

Nonfunctioning tumors of the adrenal medulla are of 2 types — neuroblastoma and ganglioneuroma. The latter may arise from mature sympathetic ganglion cells anywhere, but in all locations these tumors are benign and cause only symptoms due to their location. These occur most often in children or young adults. The neuroblastoma of the adrenal arises from the primitive neuroblast and is a highly malignant neoplasm characteristically found in infancy and childhood. It is the second most common abdominal tumor in infancy and childhood. The most common initial complaint is abdominal swelling with or without pain. Pallor, fatigue, weight loss and loss of appetite may be noted. By far the outstanding physical finding is the presence of an abdominal mass. This mass is usually firm, nodular and, although mostly on one side, may spread across the midline. The liver may or may not be enlarged and nodular. These tumors commonly metastasize and the metastases are frequently widespread to the kidneys, orbits, liver, brain and other organs with a predilection for the skeletal system. The location of the metastases is frequently the differentiating point between this tumor and the more common Wilms's tumor, since the latter more commonly spreads early to the lungs.

SPECIFIC DIAGNOSTIC METHODS

The diagnosis of all adrenal tumors must generally be suspected from the history and physical findings or it will not be made. However, these alone are not specific and one must fall back on other diagnostic adjuncts. The more important of these are tests for pheochromocytoma, urinary hormone determinations and roentgenographic examinations. These will be reviewed briefly.

Tests for Pheochromocytoma—There are 2 groups of drugs which are currently being used to help substantiate the diagnosis of pheochromocytoma: the histamine group, representing the provocative tests, and the adrenolytic group. In 1945, Roth and Kvale [6] showed that the intravenous injection of histamine produced attacks similar to spontaneous pheochromocytoma attacks. This has been used as the basis of the histamine test. To be performed properly, it should be preceded by the cold pressor test. In this latter test, after determining the basal blood pressure, the hand is placed in ice water and the blood pressure is checked every thirty seconds for seven minutes. The histamine test is then performed by again determining the basal blood pressure and then injecting .05 mg. of histamine base in .5 ml. of isotonic saline intravenously and checking the blood pressure and pulse every minute for fifteen minutes. A positive test is a response greater than the cold pressor test with a maximum reached at the end of two minutes. The average rise in histamine tests performed by Roth,[7] in patients with pheochromocytoma, has been 104/56. This test finds its greatest usefulness in those patients with paroxysmal hypertension. It is dangerous to use in the patient whose blood pressure is in the hypertensive range (over 170 systolic). Other drugs which have been used in a provocative manner are mecholyl chloride and tetraethylammonium chloride, but neither of these has given as good results as histamine.

The second group of drugs which has

proved of great value in determining the presence of pheochromocytoma is the group of so-called "adrenolytic drugs." These drugs block the effects of the circulating pressor substances and if the hypertension is due to this, the blood pressure is decreased. The 2 drugs of this type which are currently enjoying the greatest usage are regitine and piperoxan. These drugs are used in those patients whose systolic blood pressure is greater than 170. To perform the test, a needle is inserted in a vein and repeated determinations of the blood pressure are made until the pressor effect of the needle puncture has worn off. Five mg. of regitine in 1 ml. of solution is injected instantaneously and the blood pressure is recorded every thirty seconds for ten minutes. A positive result consists of a maximal depressor effect sustained for three or four minutes after the intravenous injection, and this response should exceed 35 mm. systolic and 25 mm. diastolic. At the Mayo Clinic the average fall of 19 patients with proven pheochromocytomas was 75/56.[7] It is important that these patients have no narcotics or sedatives for twenty-four to forty-eight hours prior to the test because this is the greatest cause of false positives. Antihypertensive drugs should be stopped fourteen to sixteen days before the test, because these may produce false negative results.

These tests have been of great help in making the diagnosis of pheochromocytoma but they are not infallible. It is probable that, in the not too distant future, they will be completely replaced by more direct determinations of circulating epinephrine and norepinephrine in the blood or of their excretion products in the urine. Currently values for pressor amines in the blood[6] and catechol amines in the urine[2] are being determined, but these tests are still in their infancy and are not being widely used.

Urinary Hormone Determinations—The urine contains a large number of steroids which are derived from the gonads and the adrenals. The majority of urinary steroids have a keto group at C-17 and are called 17-ketosteroids. Recently, with improvements in clinical tests, corticoids (compounds E and F, the 11-oxycorticoids and desoxycorticoids) have been found and analyzed in the urine.

The neutral 17-ketosteroids come from 2 sources, the testis and adrenal cortex. Interstitial cell tumors of the testis, masculinizing ovarian tumors, some cases of arrhenoblastoma, and most cases of Cushing's syndrome have increased excretion of urinary 17-ketosteroids. (The normal value for men is 14 mg. per twenty-four hours and in women 7 to 10 mg. per twenty-four hours.)

The methods of analysis for urinary 11-oxycorticoids and desoxycorticoids are still difficult and may be too elaborate for routine clinical use. Nevertheless, in Cushing's syndrome these are elevated above the normal values (11-oxycorticoids .01 to .005 mg. per twenty-four hours; desoxycorticosterone .1 to .2 mg. per twenty-four hours).

These values are usually, but not always, increased in cases with hyperfunction of the adrenal cortex. In addition, their determination has been some help in differentiating hyperfunction due to cortical tumor from hyperfunction due to a diffuse cortical hyperplasia. The 17-ketosteroid excretion may be high in both conditions but the higher figures usually indicate tumor. Furthermore, administration of cortisone by suppressing ACTH production tends to drop the 17-ketosteroids in cases of hyperplasia but not in tumor cases.

Roentgenographic Examination — There are several roentgenographic procedures now available to help substantiate a diagnosis of adrenal tumor or to help localize a tumor once a diagnosis is made. A scout film of the abdomen may show calcification, and planograms are helpful and should be utilized. Pyelograms may show abnormal displacement of one kidney by an overlying mass. Obviously the larger the tumor, the more likely are the pyelo-

grams to be helpful. They have been particularly helpful in our experience in the nonfunctioning cortical carcinomas. The smaller tumors usually associated with Cushing's syndrome and the pheochromocytomas are usually difficult to locate by these examinations.

Several years ago perirenal air insufflation was advocated by Cahill [1] to help with this problem, but it was not enthusiastically received and never enjoyed great popularity. This has now been replaced almost entirely by retroperitoneal air studies which have proved to be of some value. This examination is carried out by injecting 500 ml. of air into the presacral space through a spinal needle in the perineum assisted by a finger in the rectum. After the injection, the patient's head is elevated and films are taken up to two hours afterward. This examination has been helpful in localizing small tumors as well as the large ones. Pyelography and air studies may be combined without difficulty if it is felt that further information might be obtained in this manner.

The most recent roentgenographic help in localization of these tumors is found in aortography. Probably the information is more easily obtained through one of the above procedures but additional information can be obtained by translumbar aortograms. A delineation of the blood supply to the kidneys and the adrenals may help determine the presence or absence of a tumor mass. Again, this may be combined with either or both of the studies previously mentioned.

At times the diagnosis of an adrenal tumor may be suspected but the tests may fail to be conclusive. In some of these cases, particularly those which are pheochromocytoma suspects, surgical exploration is indicated as the final diagnostic maneuver.

SUMMARY

A brief classification of adrenal tumors, along with their clinical pictures, has been reviewed. These clinical data, along with certain diagnostic adjuncts such as pharmacologic tests for pheochromocytoma and urinary hormone determinations for Cushing's syndrome, should help one make a definite diagnosis in most cases. Further help in diagnosis or localization of the tumor can be obtained by the use of plain roentgenograms, planograms, intravenous and retrograde pyelograms, retroperitoneal air studies and lumbar aortography. Surgical exploration is occasionally indicated in doubtful cases to establish the diagnosis.

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THE TREATMENT OF OSTEOARTHRITIS WITH PARTICULAR REFERENCE TO THE USE OF PREDNISONE*

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Osteoarthritis, or degenerative joint disease, is one of the most common diseases afflicting mankind.[1, 2] The first pathological changes become apparent in the second decade, and by the sixth decade are almost universally present. However, only about 5 per cent of the population past the age of 50 years have sufficient symptoms to consult a physician. One of the problems confronting students of this disease is why at times it develops in young people, particularly young females, and again why, when it is almost always present in the older age groups, many go through life with practically no symptoms.

Because of its frequency, a thorough understanding of the treatment of osteoarthritis is extremely important. The objectives of treatment are to slow the progression of the disease, preserve joint function and relieve pain and stiffness[3] (Table 1).

TABLE 1
TREATMENT OF OSTEOARTHRITIS

1. Correction of postural deformities
2. Weight reduction
3. Rest
4. Heat—moist, dry, paraffin baths
5. Exercises to strengthen particular muscle groups
6. Drugs for relief of pain and stiffness:
   a) Salicylates
   b) Other analgesics—Propoxyphene
7. Intra-articular steroids—Frequently helpful
8. Oral Prednisone—for a limited period of time.

The most commonly involved joints are the knees, hips, lumbar spine, cervical spine, shoulders, and terminal interphalangeal joints of the hands. Stecher[4] has shown that involvement of the terminal interphalangeal joints may occur alone as a hereditary disease or as an accompaniment of a more widespread involvement. These Heberden's nodes are frequently more unsightly than painful, and may require no treatment, and pain, if present initially, may subside within a few weeks.

REDUCTION OF WORK LOAD OF INVOLVED JOINTS

Since the knees, hips, lumbar spine, and in a sense, the cervical spine are weight-bearing joints, and since repeated wear and tear produce trauma which hastens progression of this disease, everything possible should be done to reduce the work load of the involved joints. At times postural deformities, such as shortening of one leg, flat feet, residuals of poliomyelitis, and curvatures of the spine, add to the joint load. When possible, they should be corrected, preferably before the onset of symptoms or at least before extensive progression of the arthritic process. Involvement of the knees is a frequent presenting symptom, and in our experience is especially common in obese women. Although we all know the difficulties encountered in effecting an adequate weight reduction, these patients should be advised as to the consequences of continuing obesity, and a proper diet program advocated. Some patients have the idea that the proper thing to do is to "work out the pain and stiffness," and undertake strenuous exercise programs which actually hasten the progression of the disease. They should be advised that while it is usually helpful to move joints through a full range of motion several times daily, extensive exercises may be harmful. When indicated, they should be used with the idea of strengthening a particular muscle group, such as the quadriceps muscles, and should be done in such a manner as to minimize trauma to the already worn joints.

HEAT

The application of heat to the involved joints in the form of hot soaks, heating pads, infra-red lamps, paraffin baths, and so forth, produces an increase in the blood supply to the area and is often helpful.[5]

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A method that can be used in the home two to three times daily is generally preferable to less frequent office treatment. The use of hot soaks for the hands is helpful. For involvement of the knees, a hot tub bath provides symptomatic relief. However, if involvement is more pronounced in the lumbar or cervical spine the use of either a simple heating pad or infra-red lamp is more practical.

In the use of drug therapy the objectives of treatment should be kept in mind. The patient should be warned that even though he may obtain relatively complete relief from his pain, he should continue to avoid any undue strain to the involved joints, and should not use the drugs merely to permit him to do things that he could not do otherwise. If he abuses his joints it is quite likely that he will get along fairly well for a while but will pay for it later with increasing disability.

INTRA-ARTICULAR INJECTIONS

When there is involvement of only a few joints the intra-articular use of one of the four currently available corticosteroids may provide dramatic relief, and although the duration of effect of these medications is certainly less than two weeks, pain is relieved at times for a period of many months.

The intra-articular preparations that are currently available are hydrocortisone, hydrocortisone tertiary butyl acetate, prednisolone and prednisolone tertiary butyl acetate. While all of these are therapeutically effective, in our experience hydrocortisone tertiary butyl acetate seems to be effective over a longer period than hydrocortisone or prednisolone. Theoretically, the palliative effect of prednisolone TBA might be expected to equal that of hydrocortisone, and to exceed it, milligram for milligram in potency. However, it has not been available long enough for a thorough evaluation.

In general, the technique of intra-articular injection is relatively simple in all of the peripheral joints except the hips. The systemic effects of the steroids when used intra-articularly are minimal and there are no contraindications to repeating the injections at fairly frequent intervals. In an occasional patient marked pain for about twelve to twenty-four hours may follow injection, but it then abates and does not lessen the beneficial result.

ANALGESIC AGENTS

As analgesic agents, the salicylates remain in the forefront because of their reliability and minimal cost. If there is no gastric intolerance, aspirin, in a dose of 0.6 gm. to 1 gm. four times daily is generally effective. The combination of paraminobenzoic acid, ascorbic acid, or colchicine adds little to the effect of aspirin but does increase its cost. If uncoated aspirin tablets cause gastric distress, one may use either enteric-coated aspirin or buffered aspirin, such as Ascriptin, which is a combination of aspirin and Maalox, or enteric-coated sodium salicylates if there is no contraindication because of the sodium ion.

When the proper use of rest and salicylates fails to give adequate relief or in the occasional patient who cannot take salicylates, the use of other analgesic agents may be considered. For the past year we have been evaluating a new synthetic analgesic, D-Propoxyphene which is marketed under the trade name of Darvon.* In general, its therapeutic effect approaches that of codeine. Studies to date show no addicting properties and because of that it is not classified as a narcotic. It can be used either alone or in combination with aspirin, and promises to be a worthwhile addition to the therapeutic armamentarium.

PREDNISONE

In a study which we[6] recently completed on the comparative value of prednisone, 5 mg., four times daily, aspirin, 0.6 gm. four times daily and a placebo in osteoarthritis, 78.2 per cent had a satisfactory response to prednisone, 65.2 per cent to aspirin, and 40 per cent to a placebo. The response to the placebo was not expected, but it emphasizes the advantage of using adequate controls in the

* Eli Lilly and Company, Indianapolis, Indiana.

study of a chronic disease such as osteoarthritis. The response to aspirin was better and prednisone was still more effective.

There have been very few studies on the effect of the adrenal steroids when administered parenterally for osteoarthritis and certainly not every patient should be given prednisone or one of the other steroids.[7-11] While their anti-inflammatory effect is desirable and probably responsible for their therapeutic usefulness, undesirable systemic effects such as sodium and chloride retention, an increased incidence of peptic ulceration, and increased protein catabolism have limited the usefulness of the adrenal steroids.

Prednisone and prednisolone possess one advantage over the other steroids in that they do not produce the degree of sodium and chloride retention and potassium depletion that is such a problem with the other preparations. However, their therapeutic effect is no greater and other side effects may occur.

In the treatment of osteoarthritis, one must consider that it is a chronic disease and that the anatomical joint changes are permanent. This limits the degree of improvement that can be expected. However, when the arthritic process is relatively mild, it may be satisfactorily controlled by limitation of activities, physical therapy, aspirin, or one of the other salicylates, and possibly one of the intra-articular steroids. Ordinarily, all of these measures should be tried before considering the use of prednisone or one of the other steroids orally. However, the course of the disease is variable and at times exacerbations occur and it is a problem to give the patient adequate relief. In such instances, one may be justified in considering the use of prednisone for a short period of time. After one or two weeks of therapy the symptoms may subside and it will then be possible to revert to the more conservative measures outlined above for long term management.

In our study, there were no serious complications from the use of prednisone, but none of the patients received this drug for more than one month.

SURGERY

The surgical approach is generally reserved for those who fail to respond to a medical regime, and is limited primarily to knee and hip involvement.[12] In treatment of hip involvement, various types of prostheses, arthrodesis, and denervation of the joint capsule have been advocated. However, none of these is entirely satisfactory. For osteoarthritis of the knees, some orthopedists advocate removing the patella [13] and cleaning up the frayed cartilage and removing any loose bodies. Such procedures necessitate a long period of convalescence and patients are frequently reluctant to accept them.

SUMMARY

In summary, the objectives of treatment are to limit the progression of the disease, preserve joint function, and relieve pain and stiffness. This involves a comprehensive treatment program including the use of rest, heat, and properly selected exercises and salicylates. In some instances, intra-articular steroids are beneficial, and they are also effective when given parenterally. Because of their side effects, their long term parenteral use in osteoarthritis is not advocated, but they may be used for short periods of time if more acute symptoms occur. In the use of drug therapy, it should be remembered that the desired effects of treatment are to delay progress of the disease as well as to relieve joint pain.

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EYE SIGNS OF HEAD INJURY *

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Proper evaluation of the eye signs of head injury is of twofold importance: First, at least one of the signs enables us to determine those cases which must be referred to our neurosurgical colleagues immediately; second, the consideration of the eye signs helps us in determining prognosis and disability from head injury.

I. LIDS

A. N VII PARALYSIS

As we see the head injury patient lying in bed, we will notice whether he is able to close both eyes. With a nuclear to peripheral type of facial nerve (N.VII) injury, the lids of that eye cannot be closed, because of interference of nerve supply to the orbicularis oculi. Also, the ipsilateral labial commissure cannot be retracted to "show the teeth" on that side. Most of these traumatic paralyses are peripheral, and are caused by fracture from the temporal region, crossing the middle ear and the petrous pyramid.[1] The facial paralyses which occur immediately after injury are usually complete and peripheral. If they are nuclear, the VI nerve to the lateral rectus muscle of the eye is also usually involved, because of the proximity of the fibers, as is seen by an examination of a lateral drawing of the brain stem.

If the facial nerve lesion is supranuclear, then the patient can close the eyes, because of the bilateral upper motor neuron (corticobulbar) supply to the upper part of the facial nerve nucleus. However, there will be flattening of the nasolabial fold on the side opposite the lesion, and the labial commissure cannot be voluntarily retracted, because of only crossed corticobulbar or upper motor neuron fibers to the portion of the facial nucleus that supplies the lower facial muscles.

In a series of 1550 cases of head injury, Turner found 20 per cent showed facial nerve injury. (It is therefore important to keep in mind methods of protecting the thus exposed cornea: lubrication with ointment; 1 per cent methyl cellulose; an airtight shield; or sewing the lids together.)

B. PTOSIS

If the patient exhibits a drooping or ptosis of an eyelid, we must differentiate third nerve injury, from Horner's syndrome.

In third nerve injury, the powerful levator palpebrae muscle is paralyzed, and allows practically a complete ptosis on that side of the lesion; whereas in Horner's syndrome, from interference with the sympathetic nerve supply, the small band of smooth muscle (Muller's allows only a millimeter or so of ptosis. Associated with the small degree of ptosis are miosis or small pupil (from interference with the radial dilator fibers of the iris), anhydrosis, and (relative) enophthalmos.

Differentiation of the miosis will be considered later under discussion of pupil.

C. DIFFERENTIATION "BLACK EYE" AND FRACTURE OF SKULL

Another point of interest about the lids is the differentiation of the common "black eye" from fracture of the base of the skull, with its hemorrhage seeping forward into the lids.

The septum orbitale is the "fire wall of the orbit," presenting a definite obstacle or barrier to inflammatory or neoplastic diseases arising on either side of it, and pre-

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vents hemorrhagic extravasations from progressing farther anteriorly, from their point of origin posteriorly. Thus, this thin membrane of connective and elastic tissue, attached to the thickened line of periosteum around the orbital rim, prevents hemorrhage from seeping into the superficial tissues of the lid. Therefore, in fracture of the base of the skull, hemorrhages are limited to the orbital margins, but in "black eye" the hemorrhages are not limited.

In the "black eye", the conjunctiva is immediately [1] red, the conjunctival hemorrhage moves, and shows a posterior limit to the extravasation, and the superior tarsus is usually the first to show the marked redness. In fracture of the skull base, however, the conjunctiva becomes purplish after several days, the hemorrhage on the eyeball does not move, and does not show a posterior limit as the handle of the fan is toward the iris, and the lid discoloration first shows in the midlower lid. In black eye there is relatively more edema than blood, whereas in fracture of the base the edema and blood correspond.

II. CONDITION OF PUPILS

Of utmost importance is the condition of the pupils in the head injury patient.

If the pupil is dilated on one side, it may be of grave importance from injury to the brain and the parasympathetic nerve supply to the constrictor of the pupil, or it may be dilated from other causes.

Of great etiologic significance in the dilated pupil from brain injury is the tentorial gap, or incisura. This is the space enclosed by the free border of the tentorium cerebelli. The tentorium is the tentlike shelving of dura which separates the cerebellar lobes below from the occipital lobes above. The attached border begins at the occipital protuberance, includes the lateral sinus as it runs forward, then the superior petrosal sinuses, where the attached border swings inward along the superior angle of the temporal bone, to be attached to the posterior clinoid process.[2] The *free* border of the tentorium, with which we are especially concerned, begins at its forward attachment to the *anterior* clinoid process, and encloses the tentorial hiatus or gap as it passes backward to encircle the midbrain.

As the oculomotor nerve fibers leave the midbrain, they course through the crotch of the fork [3] made by the superior cerebellar artery below, and the posterior cerebral artery above, as they are given off the basilar artery. The nerve passes forward, through the interpeduncular cistern, and to the superior portion of the cavernous sinus, which it then enters.

The posterior cerebral arteries, which are the paired terminal branches of the basilar artery, run forward and laterally, in the tentorial gap, and then backward around the cerebral peduncles of the midbrain, coursing between the peduncles, and the uncus of the temporal lobe. These posterior cerebral arteries then pass over the free border of the tentorium, to course above the tentorium, to the area they supply. Two important areas supplied are the striate or visual area of the cortex of the occipital lobe, and part of the oculomotor nuclei.

Postmortem examinations of normal brains show that the uncus, or hook-like portion of the hippocampal gyrus of the temporal lobe, project as much as 5 mm. below the free margin of the tentorium, with variable degrees of grooving.

However, when there is increased pressure above the tentorium, the hippocampal gyrus is forced as much as one or two centimeters below the tentorial margin, with deeper grooving.[4] This herniation is caused by rapidly increasing supratentorial pressure, which in turn may be caused by: rapidly expanding tumors (such as the one reported by Echols [5]); abscesses; cerebral edema from injury; cardiovascular and renal conditions; aneurysms, and especially, hemorrhages.

With the increasing supratentorial pressure from epidural and subdural hemorrhages, as the uncinate gyrus and brainstem are forced downward, several important changes occur: (a) The oculomotor nerve is at first compressed from above by the downward pressing temporal lobe uncus.[6-8] The more superiorly placed (and possibly more sensitive) pupilloconstrictor fibers are first affected, before the fibers to the levator and extraocular muscles. An

initial miosis may or may not result from irritation, but this rapidly gives way, even within a few minutes, to the paralytic mydriasis, within one to twelve hours. This mydriasis may also be caused by aneurysms of the adjacent vessels. (b) The oculomotor nerve then becomes angulated acutely by the posterior cerebral arteries, and stretched, as the pons becomes displaced downward by the herniating cerebral mass. The basilar artery, which is tethered to the pons by short branches, moves distally with the pons and the elongation of the mesencephalon, carrying with it its origin of the posterior cerebral artery. Therefore, each posterior cerebral artery must ascend more acutely to reach the tentorial edge, and therefore drags more on the superior surface of each oculomotor nerve. (c) The posterior cerebral arteries are forced [4] against the sharp tentorial rim, thus giving anoxia and ischemia and edema of the occipital lobe. A homonymous hemianopsia results. These field defects and pupillary changes are reversible if the pressure is relieved. However, actual infarction of the artery occurs with prolonged pressure on the vessel, and thus destruction of the occipital cortex results.

Further compression and dislocation of the midbrain cause actual closure of the aqueduct of Sylvius. Edema and hemorrhages in the brain stem so embarrass the vital centers that death results.

In addition to brain swelling with herniation of the temporal lobe, other causes of dilated pupil to be considered are: physiological, but here the pupil reacts normally; direct injury to the eye, but the pupil is often irregular; unilateral blindness, but there is still reaction to indirect light; cycloplegia; tonic pupil of Adie, but there is miosis with prolonged convergence, and mecholyl causes narrowing; and quadrigeminal plate lesions (usually tumors).

As previously considered with Horner's syndrome, a small pupil may make the opposite pupil appear larger. The larger pupil will react well if it be physiologic, but not if from paralysis of the pupilloconstrictor. Consideration of small pupil makes us consider one that is congenitally so, either physiologic or by absence of dilator fibers of the iris. Acquired miosis may be from miotics, old inflammatory residue, irritative from meningitis or pontine hemorrhages, or the sympathetic paresis of Horner's syndrome.

If a patient is seen immediately after an injury, and has a dilated and fixed pupil on one side, then it is usually due to a laceration of the third nerve or its nucleus, and there is usually a complete ptosis as part of a complete nerve injury. However, a dilation occurring about an hour or more later is usually without ptosis early, and is due to hemorrhage.

III. CORNEAL REFLEX

The corneal reflex may be tested by touching the cornea with a wisp of cotton. Of course the eyelids will close if the reflex pathways from afferent branches of the ophthalmic division of NV are intact, along with the central connections, and the efferent pathway through the facial nerve nucleus and the peripheral NVII, to the orbicularis oculi.

There has been some debate as to whether the cornea itself has its center for touch in the mesencephalic nucleus of NV as do the lids, and pain and temperature in the spinal tract and nucleus of NV, as do also the lids. However, evidence presented by Cogan indicates that *all* sensations of the cornea, including touch, are centered in the spinal nucleus.

IV. CONJUGATE DEVIATION

In general, even in the unconscious patient, determine if possible whether the eyes seem to rove well in all directions, or whether there is definite limitation in one direction.

If there be conjugate deviation of the eyes to one side, then brain involvement is indicated, but the exact site can be one of several places.

Stimulation of the voluntary eye motor center of the frontal lobe (posterior part of the middle frontal convolution) incites conjugate deviation to the side opposite the lesion, and opening and closing of the lids. Stimulation of the occipital visual areas of the cortex (especially the angular gyrus 39, and the peristriate area 19 bordering the anterior occipital lobe), and the association

paths between them and the eye motor area of the frontal lobe, produce conjugate but usually transient, deviation of the eyes to the side opposite the lesion; whereas the afore-mentioned lesions of the frontal area are a little longer in duration. In the frontal eye motor area, a lesion may also involve the adjacent area for head movement, located above, or the writing area situated posteriorly in the precentral area of the frontal lobe, or the motor speech area of Broca which lies just below the eye motor area. (Thus, conjugate deviation may be associated with motor agraphia and aphasia, as well as a deviation of the head).

Fibers from the voluntary eye motor area as corticobulbar or aberrant pyramidal fibers, and pass downward through the corona radiata with fibers from other motor areas. These corticobulbar fibers pass through the genu, or knee, of the internal capsule, and descend as part of the cerebral peduncle into the brain stem. These fibers to the eye motor nuclei pass chiefly to the opposite side, but the exact site of crossing is debatable.

This explains why stimulating lesions of the occipital visual or the frontal eye motor areas, or the knee of the internal capsule, or the cerebral peduncle, give conjugate deviation of the eyes to the opposite side, and possibly to the same side if the irritative lesion be in the pons; whereas in destructive lesions of the upper motor neuron, the conjugate deviation is to the same side as the lesion, and probably to the opposite side if the destructive lesion be in the pons. Lesions of the pons may also implicate the facial nucleus or nerve, and even the pyramidal tract and medial fillet to give contralateral hemiplegia and hemianesthesia of the body.

We feel that there is great difficulty in a rapidly changing clinical course, in determining whether the lesion is changing from irritative to destructive, or whether new areas are becoming involved. Therefore, we feel that the presence of conjugate deviation of the eyes is of value in denoting injury to the brain, but do not feel that it is of great localizing value.

V. PARALYSIS OF EXTRAOCULAR MUSCLES

All the nerves to the extraocular muscles may be paralyzed by a lesion at the apex of the orbit, where nerves III, IV, and VI enter the orbit through the superior orbital fissure as so well shown by Fralick, McCotter and Henderson.[9] Ptosis is apt to be present, as part of the paralysis of the other elements of the third nerve. However, Walsh [10] states that this is an extremely rare occurrence. Pfeiffer studied a large series of skulls in the Army Medical Museum, and found involvement of the optic canal to be very rare.

Paralyses of various nerves of the extraocular muscles may occur from subarachnoid hemorrhages, which are quite common at the base of the brain. The nerves are involved by hemorrhages into the interpeduncular and chiasmatic cisterns. The III nerve is especially vulnerable here, but the trochlear nerve is only rarely involved.

Although Lyle states that the VI nerve is more frequently involved in injury than the III nerve, Turner only found each of the two nerves involved in 6 per cent of the 1550 cases of head injury, while the VII nerve was involved in 20 per cent. Although the VI nerve is especially vulnerable to injury as it ascends the brain stem and turns forward through Dorello's canal, over the apex of the temporal bone, it may also show weakness from increased intracranial pressure, and is not of much localizing value. The cases of VI nerve injury from increased intracranial pressure are usually delayed.

VI. NYSTAGMUS

Nystagmus may be noted in head injury cases, but is not of great localizing value. Although it has been ascribed to labyrinthine injuries, it can result from injury to the central connections of the vestibular nerve in the brain stem, or even to secondary vestibular connections, and Rucker's excellent discussion [11] of nystagmus emphasizes that nystagmus is not of good localizing value. However, Lindsay [12] has cautioned that in the late head injury cases, elicitation of positional nystagmus by postural tests may be the only and valuable

objective sign of the subjective complaint of vertigo.

VII. OPTIC NERVE

A. VISION AND FIELDS

Thus far we have considered eye signs of involvement of the third, fourth, fifth, sixth and seventh cranial, and the sympathetic nerves, in the head injury case. Now let us consider the second cranial nerve.

If the patient be conscious, it is of great importance to early assess the patient's vision, not only for clinical, but also before the compensible aspects of the case loom too large. If it is impractical to use a visual acuity chart, then it is at least valuable and easy to determine ability to count fingers at specified distances.

Although detailed perimetric studies of the visual field are usually not feasible in the acute head trauma case, confrontation visual fields at least detect hemianopsia. Blind spots will be considered later.

B. PAPILLEDEMA

Determination of presence of papilledema is important. Although many authors state that papilledema usually does not develop for as long as three to seven days after acute head trauma, it has been observed in as little as twenty-four hours.

It is convenient to divide the findings of papilledema into three stages. In the early stages (first few days) there may be but few findings, such as increased redness of the disc, blurring of margins, movement of small capillaries on and near the disc with parallax, filling of the physiologic cup, development of gray streaks along the emergent vessels, and engorgement of the retinal veins. (Walsh [10] finds this latter development of considerable importance in the early stage). In the later or second stage (seven to ten days): the entire disc outline becomes blurred, the physiologic cup becomes completely filled, and there is measurable swelling of the disc (2D). In the more advanced or third stage, about ten days onward, the disc is red like the surrounding retina, the swelling has increased from 5 to 9 diopters, the edges of the disc overhang the retina mushroom-like, the engorgement of the retinal veins is associated with hemorrhage, and there is a macular fan toward the disc.

From the above, it is seen that progression toward papilledema may be gradual, so it is important that we record in detail, from each examination, all those characteristics that may be of value in detecting the progressive changes leading to a diagnosis of papilledema.

Disc characteristics may be recorded by using Elschnig's types (Bothman) [13], or we may follow a recording system somewhat as suggested by Berens.[14] Disc characteristics should be recorded as to color, borders, presence and degree of physiologic depression, and as to whether the gray stippling of the lamina cribrosa is markedly distinct in the bottom of the physiologic depression, distinct, indistinct or not visible. Presence or absence of tufts of grayish tissue about the emergent vessels should be noted, as should dilatation of the retinal veins, and the presence of hemorrhages and exudates.

A number of conditions must be differentiated from papilledema.[15] Of the four usually considered developmental and congenital conditions, pseudopapilledema presents the discs swollen and elevated, but not containing edematous tissue that disturbs retinal physiology, and although the veins may be full and tortuous, they exhibit more graceful curves than do the vessels in papilledema. Pseudopapilledema is usually associated with hypermetropia. Hyaline bodies or drusen of the optic nerve head, a second developmental condition, are easily identified if the coin-like bodies are readily visible if they are near the surface of the disc, but differentiation from papilledema may be very difficult if the yellowish amorphous material is buried beneath the surface, and we should not hesitate to call on our colleagues for the findings of any previous examinations. Hemorrhages are not present with hyaline bodies. Myopic changes about the disc are a third developmental condition in differential diagnosis. However, there are no hemorrhages or venous congestion, and no enlargement of the blind spot unless there is a scleral crescent or posterior staphyloma. Medullated nerve fibers are a fourth congenital condition, whose characteristic whitish brush-like striations, usually coming off the upper and

lower borders of the disc, may be confused with papilledema.

Of the inflammatory conditions, optic neuritis is often most difficult to differentiate from papilledema by fundus picture alone. Both conditions show edema, blurring of borders, congestion, and hemorrhages and exudates. However, optic neuritis is usually unilateral, presents marked loss of central acuity, tenderness and pain on motion of the eyeball (because of intimacy of the optic nerve sheath and the muscle cone superonasally), loss of vision for red, and cecocentral field defects rather than enlargement of the blindspot. Chamlin and Davidoff state that the normal blindspot is 5.5 degrees to 7.5 degrees wide, and 7.5 to 9.5 degrees long, as determined by 2 mm. white spherical test object at 2 meters distance. In papilledema the blindspot enlarges, and in their case of acoustic nerve tumor, an increase in size of the blindspot was noted at least three weeks before the appearance of definite ophthalmoscopic evidence of papilledema. These authors state that they have never seen papilledema with a completely normal blind spot. However, Payne [16] states that he cannot confirm the great value assigned by the authors to enlargement of the blindspot in incipient papilledema. Walsh [10] and King [17] quote Hill that if blindspots continue to show enlargement twelve hours after head trauma, then edema of the brain is present, whether or not there are ophthalmoscopically visible changes.

Choroiditis juxtapapillaris (Jensen) is an inflammatory condition of the choroid and retina near the disc, which is usually unilateral, eventually shows pigment migration there, and field changes of blindspot enlargement, cecocentral defect, or nerve fiber bundle defect.

With vascular changes, papilledema may be suggested, or actually present, in hypertensive neuroretinopathy, peripapillary vascular sclerosis, and thrombosis of the central retinal vein. However, the vascular changes and hemorrhages are usually seen peripherally, also.

VIII. ARTERIOVENOUS FISTULA

Arteriovenous fistula may develop in the cavernous sinus, from injury on the side of the head or about the lateral wall of the orbit. The etiologic trauma may be fairly mild, and not associated with loss of consciousness, but there is often a fracture through the cavernous sinus. The patient hears a buzzing noise, and the physician hears a bruit by placing a stethoscope over the eye (although it might possibly be on the side opposite the lesion, presumably because of abnormal communication between the cavernous sinuses.) There is engorgement of the ocular vessels, and a pulsating exophthalmos. There may be incomplete extraocular muscle palsies, or late papilledema. About 10 per cent of the cases are bilateral.

SUMMARY

Numerous eye signs of head injury have been discussed. Some of these signs are of value in indicating the degree of severity of the injury, but are not of great localizing value. The importance of the dilated pupil is stressed as a danger sign of herniation of the hippocampal gyrus of the temporal lobe. Also stressed is the importance of detailed recording of fundus characteristics required in the diagnosis of papilledema.

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DISCUSSION

Dr. George M. Haik (New Orleans): If I were to select the most important point which Dr. Harms has made in this excellent paper dealing with the eye signs associated with head injuries, I think my choice would be his insistence on the cooperation necessary between ophthalmologists and neurosurgeons in this type of trauma if the best interests of the patient are to be served. There has always been a theoretical appreciation of the necessity for such cooperation. Practically, however, it has sometimes been less than complete.

Combined ophthalmologic and neurosurgical care of battle-incurred head injuries saved both sight and lives during the second World War and during the Korean conflict. It is important that such combined care be the practice in civilian life, for two reasons. The first is that in this highly mechanized age we are managing to kill thousands of persons each year in automobile and other accidents and are injuring millions of others. Head injuries are a large component of the total. The second reason is that this is an age in which all of us live in the shadow of a possible mass disaster, in which, if it comes, there will be thousands of head injuries to be cared for.

Dr. Harms' paper is an orderly consideration of the diagnostic, therapeutic and prognostic significance of eye signs in head injuries, beginning with the state of the eyelids and ending with the possible damage to structures of the internal eye. The limited time allowed him for his presentation scarcely permitted him to hint at what his paper contains. As one who has had the privilege of reading it in full, I assure you that it will repay not one reading but several.

I would like to emphasize one or two points which impressed me as I read this paper. The first is the sound common sense of testing vision in conscious patients, not only for the clinical value of the observation but, as Dr. Harms put it bluntly, before the compensation aspects of the case loom too large. He is quite correct. We live in a material world, and these aspects cannot be ignored from the standpoint of either the injured man or the employer or insurance company responsible for compensating him for his injury. It may not be practical at the time of injury to use a visual acuity chart but it is both possible and useful to determine the ability to count fingers at a specified distance. Also, although detailed perimetric studies of the visual fields are usually not practical, confrontation fields provide a simple method of detecting hemaniopsia, while simple questioning will determine the existence of such complaints as double vision.

Dr. Harms also emphasizes the importance of tracing and recording the development of papilledema. As he says, the progression may be so gradual that without careful examination and equally careful recording, the slight but highly significant changes in the fundus may be overlooked until they are far advanced. He has provided a comprehensive list of the signs which should be sought for and recorded.

Finally, Dr. Harms has pointed out that while in a severe head injury, which is potentially lethal, most of the attention must be directed toward the injury, it takes little additional time and effort to protect the eye. When the facial nerve, as he says, is injured in perhaps 20 per cent of all cases, the cornea is likely to be exposed in a certain proportion of these. If so, it should be protected by some simple method, including, if necessary, sewing the lids together.

This is a paper in which there is really nothing left to discuss. In his comprehensive presentation, Dr. Harms has said it all already.

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TREATMENT OF TUBERCULOSIS IN MAN

The treatment of pulmonary tuberculosis in man, so far as effective drugs are concerned, began in 1947 when streptomycin was made available for clinical use. Following this, after a brief period, two other drugs—isoniazid and aminosalicylic acid—also became available.

In the ten succeeding years, the treatment of pulmonary tuberculosis underwent many changes. Shortly after these three drugs came into use cooperative studies in the chemotherapy of tuberculosis were undertaken by the Veterans Administration - Armed Forces group. The Council on Drugs of the American Medical Association issued a highly informative report by Raleigh and Steele [1] on the 16th Conference of this group held in February 1957. The observations and findings represented in the report are valuable to the practitioner, upon whom great responsibility for directing various phases of the treatment of tuberculosis now rests. This is important as it is estimated that approximately 56 million Americans are still harboring tubercle bacilli.

Earlier reports from this group showed that the three drugs active in tuberculosis should not be used singly, but always two at a time and that treatment should be continuous for many months. Their comparative efficacy was measured by the incidence of bacteriological conversion by culture, significant improvement by x-ray, and closure of all visible cavities. Judged by these standards, the measurable superiority of isoniazid plus aminosalicylic acid in patients with moderately advanced and far advanced tuberculosis after four, eight, and twelve months of therapy was demonstrated.

However, a definite limit to the absolute efficacy of the three combinations of these three drugs has been shown. Cavity closure was achieved after eight months of therapy in less than 40 per cent, and after twelve months of therapy in only about 50 per cent. The chances of closure were greater in the smaller cavities, and best in those below 2 cm. in diameter. Cavities persisting through twelve months of therapy rarely closed without resort to new drugs, or surgery, or both. Among patients whose sputum and gastric cultures had become consistently negative, even though the cavity persisted, continued treatment resulted in a closure in only 26

[1] Raleigh, James W., and Steele, John D.: Recent developments in the treatment of tuberculosis in man, J.A.M.A. 106:921 (Feb. 22) 1958.

per cent, and at the same time, 10 per cent of the group became worse.

It was felt that at the end of eight or twelve months continued treatment was likely to result in disappointment, and that at this time change of drugs or surgical intervention was to be considered. It was reported that among the newer drugs in use 500 mg. cycloserine with 300 mg. of isoniazid on small numbers of cases has been observed to be equivalent in effectiveness to isoniazid with aminosalicylic acid. Toxic effects were reported from the use of larger doses of cycloserine. Pyrazinamide was reported as causing toxic effects on the liver, and also, causing hyperuricemia.

It is also stated that in 30 to 60 per cent of patients isoniazid is converted into a form that is biologically inactive against the tubercle bacillus. This inactivity cannot be corrected by increasing the dose of isoniazid.

If cultural and radiographic evidences are indicative of threatened failure of therapy after eight to twelve months, the prospects of thoracic surgery with a change to a new drug are encouraging. In 5,054 pulmonary resections for tuberculosis in approximately 40 Veterans Administration hospitals, between 1952 and 1956, the surgical mortality was as follows: 15 per cent in 259 pneumonectomies; 3 per cent in 1800 lobectomies; 1 per cent in 2254 segmental resections; and zero in 732 subsegmental resections. The morbidity and mortality were influenced by the extent of the disease, presence or absence of tubercle bacilli in the sputum at the time of operation, resistance of these organisms to the drug in use at the time of operation, and the availability of additional drugs to replace those to which the tubercle bacilli were resistant.

Reports of a small, controlled study as to the effect of liberal ambulation versus bed rest indicated that there was no significant difference in the two study groups with respect to x-ray improvement, achievement of negative sputum cultures, and cavity closure. It was noted, however, that many of the supposedly ambulatory group were not out of bed and ambulatory even when they were asymptomatic.

It seems apparent, therefore, in this report that the efficacy of chemotherapy in tuberculosis must be supplemented in unfavorable cases by thoracic surgery, and that this should be considered after eight to twelve months of consistent treatment when cavities persist.

With effective means of treating pulmonary tuberculosis such as these, the ultimate control of the disease becomes more than ever a matter of case-finding.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

H. ASHTON THOMAS, M.D.
President
1957 - 1958

Dr. Ashton Thomas, our President during the past year, has had a very busy administration and has been ever watchful for any evidence of possible problems which might be detrimental to organized medicine, and has supported all measures which were for the good of our Society and its members. Dr. Thomas has conducted the affairs of the Society, always keeping in mind what was best for its members and the Society. While Dr. Thomas has had individual ideas regarding the problems presented for consideration, he has always welcomed suggestions and advice from other members of the Society.

Our President represented the profession at many parish, district and national organizations and has always exerted every effort to create good public relations between the lay public and our profession.

Dr. Thomas was born in New Orleans on July 9, 1903, and received his education in the public schools of New Orleans. His premedical education was at Tulane and after teaching in the Department of Anatomy at Tulane, 1925-1927, reentered the Medical Department of Tulane and received his M.D. degree in 1929.

After graduation, Dr. Thomas served a one years' internship and his residency in Ear, Nose and Throat at the Charity Hospital, New Orleans, 1930-1933. He entered private practice in 1933, limiting his work to otolaryngology.

Due to a physical disability, Dr. Thomas was rejected for military service in 1941, however, he has served for many years as a member of the Selective Service Commission for Physicians.

Dr. Thomas has been a member of the Faculty of the Louisiana State University School of Medicine since its organization, some twenty-five years ago, and is Clinical Professor, Department of Otolaryngology at this time.

He has served as Chairman of the Council and on many committees of the State Society, and was a charter member of The New Orleans Graduate Medical Assembly. He has been affiliated with Public Health, having served as a member of the New Orleans Board of Health and the Coroner's Commission for many years.

Dr. Thomas is Past-President of the Orleans Parish Medical Society and the Mercy Hospital Staff and is now one of the chief surgeons on the staff of this hospital. He holds fellowship in the American Academy of Otolaryngology and Ophthalmology, and is a charter member of the Board of the American Rhinologic Society. He is a senior visiting surgeon, Charity Hospital of New Orleans, and assistant chief otolaryngologist for the Illinois Central Railroad.

He also enjoys membership in the Or-

leans Parish Medical Society, Louisiana State Medical Society, New Orleans Academy of Otolaryngology, Southern and American Medical Associations, Association of Railway Surgeons, American Rhinologic Society, and the Louisiana-Mississippi Ophthalmological and Otolaryngological Society.

The members of the Louisiana State Medical Society are indebted to Dr. Thomas for his loyal and efficient services during the past year and it is sincerely hoped that he will continue his enthusiastic interest in organized medicine and will always feel free to wisely advise and counsel us.

A REVIEW OF TREATMENT SERVICES FOR ALCOHOLICS IN A SEPARATE WARD OF A LOUISIANA STATE MENTAL HOSPITAL

A twenty-five bed ward for white male alcoholics of East Louisiana State Hospital at Jackson is a pilot program to determine the feasibility of giving individual and group treatment to a screened segment of the estimated 65,000 alcoholics in Louisiana. The treatment services of the separate ward will not only be directed toward the medical aspects of the illness of alcoholism but also the often neglected social aspects. Several factors prompted the undertaking of this constructive approach of separating non-psychotic alcoholics from over 4000 institutional patients with varying degrees of mental complications.

Influencing Factors

Some of the influencing factors toward establishing this separate ward for alcoholics in a mental hospital setting include:

(1) The Louisiana Legislature's recognition that alcoholism should be treated as an illness prompted the State Department of Hospitals to accept alcoholism as a part of the mental health services.

(2) Experience and research have shown that some cases of alcoholism require extensive medical, psychiatric, and social treatment.

(3) Such a setting will afford the opportunities for a defined approach to the patient's emotional structure. Group living of patients who have similar experiences will produce the beginning of an awareness of belonging and a feeling of acceptance.

(4) Throughout Louisiana members of Alcoholics Anonymous not only voiced a favorable opinion on a program of this type, but also expressed a desire to sponsor willing patients in their local AA Groups following their discharge from the alcoholism services of East Louisiana State Hospital.

(5) Several state-wide community educational programs on alcoholism were held in 1957 as a preparatory measure for this new approach. Such programs influenced this extension of services for alcoholics, especially the plan for follow-up services.

(6) A full time professional worker in the field of alcoholism on the Department's Staff to assist in coordinating in-patient and follow-up services was an influencing factor in initial and long range planning.

(7) This program will give additional beds to mental patients in the other two State Mental Hospitals and at the same time give the alcoholic more opportunities for total rehabilitation.

Admission Procedures

Primarily, patients will be admitted to the Alcoholic Ward through legal commitment by one of the 64 parish coroners of the State. Under special circumstances, patients may volunteer for admission without court proceedings.

All white male alcoholics will initially be housed in the separate alcoholic ward for screening purposes. Alcoholics with complications, and psychotics and mental deficients, will not be retained on the separate alcoholism services. An accepted patient will have demonstrated some degree of **positive motivation,** or at least a degree of **potential motivation** to arrest his illness by cooperating with the therapists.

Therapeutic Staff and Procedures

A minimum staff has been selected to be known as "the treatment team"; it will include a medical director, an assistant psychologist, and three male attendants, plus the part-time group to include two institution counselors, a psychiatric social worker, one registered nurse, two chaplains, one steno-clerk and a psychiatrist. In addition, a vocational counselor of the State Vocational Rehabilitation Division will give necessary services, and members of outside Alcoholics Anonymous Groups will conduct a weekly AA meeting on the ward.

No set concepts for therapeutic procedures are prescribed, but some of the defined services give a broad picture of the planned treatment process. Following evaluation of the patient's willingness to cooperate, and diagnosis of the type of alcoholism involved, a course of treatment will be determined. The types of treatment will include physical therapy, psychiatric and psychological evaluation, individual counseling or short term case work, group therapy and a concerted program of Alcoholics Anonymous.

An extensive effort is to be given to treatment on the social level which is aimed not only at the patient but also at the environment in which he lives. Recognizing the obvious limitation of treatment which is carried on solely in an institution, and that such therapy can not be more than a

first and incomplete step in recovery, each willing patient will have an AA sponsor in his community prior to discharge from the hospital. Additional community resources, such as the agencies of Vocational Rehabilitation Division, Employment Security Division, selected State Guidance Centers, and other agencies, will be used to assist the recovering alcoholic in adjusting to his environment.

Pitfalls to Avoid

There are several pitfalls to avoid in starting a specialized treatment program for alcoholics. Experience with special programs for the alcoholic in four of our State General Hospitals, the teachings of the Yale University Center of Alcohol Studies, and the development of this separate ward at East Louisiana State Hospital have pointed out the following indicated pitfalls:

(1) To ignore the community's lack of understanding, and the existing common misconceptions on the nature and extent of alcoholism will work an undue hardship on the success of this type program. As Abraham Lincoln once said, "With public sentiment one can do anything, without it, one can do nothing." The need for community education on alcoholism and its associated problems has to be a continuous function if a program of this type is to be of value to the patient and the environment.

(2) Selection of employees to staff such an alcoholic ward can prove to be one of the program's greatest stumbling blocks for successful services. Each employee must have an understanding of the problems of the alcoholic and the ability to reflect this understanding in a working relationship.

(3) Avoid public conceptions that such an alcoholic ward in a mental hospital is the ideal setting to refer and treat all types of alcoholics. It has been shown not only in Louisiana but also nationwide that a great number of the alcoholic population can be handled and treated in planned programs in general hospitals and out-patient clinics.

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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Indepencence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SOCIAL SECURITY FOOTNOTES

Since Bismarck introduced socialized medicine in Germany three quarters of a century ago, the threat of socialized medicine through the extension of so-called social insurance has been ever-present in Western civilization. One nation after another has succumbed to the drive to extend the compulsory system of taxation called social insurance to finance a vast program of medical and hospital care for taxpayers and nontaxpayers. The history of developments in this field in foreign countries should alert the medical profession to the usual consequence of federal social security program.

The late Robert A. Taft classified the Social Security Act as our greatest single step toward socialism.

Every argument which has ever been used to support social security can be used with equal validity to support socialized medicine by changing a few words. If you ask for the one, prepare to get both. It is planned that way.

Those who sponsor social security regard professionals as a source of income and admit that most of them will never claim any benefits. Professional people are to be the source of funds to pay the "benefits" of others.

To support socialistic practices in regard to retirement funds requires that, for the sake of consistency, socialized medicine also be supported. If one believes that the federal government should

tax everyone to provide an income for each upon retirement or disability, one must also believe that the same government should, with equal propriety, tax everyone to provide medical care for all!

How high will the social security tax go? No one knows. But in South America some countries are paying tax rates as high as 25% of payroll. In France, the tax rate is 35% of much of their payroll and is one of the principle reasons for the failure of the French economy to make a postwar comeback.

PROGRAM
SCIENTIFIC SESSION
LOUISIANA HEART ASSOCIATION

an Affiliate of the American Heart Association

MAJESTIC HOTEL
LAKE CHARLES, LOUISIANA

May 17, 1958

9:00 A. M.

Myocardial Infarction—Emergency Treatment

Harold P. Chastant, M. D., Lafayette, Louisiana

9:30 A. M.

Management of Angina Pectoris

Cyril T. Yancey, M. D., Monroe, Louisiana

10:00 A. M.

Recess

10:15 A. M.

Practical Aspects of the Present Controversies on Etiology of Atherosclerosis.

Francis L. Chamberlain, M. D., San Francisco, California—President-elect, American Heart Association

11:15 A. M.

Panel Discussion: Coronary Disease

Drs. Chamberlain, Chastant, Yancey and Don Chapman, M. D., Houston, Texas

12:15 P. M.

Recess

2:00 P. M.

Use of Drugs in Cardiac Arrhythmias

John W. Deming, M. D., Alexandria, Louisiana

2:30 P. M.

Emphysema and Cor Pulmonale

Clay A. Waggenspack, Jr., M. D., Baton Rouge, Louisiana

3:00 P. M.

Recess

3:15 P. M.

Pericarditis

Don Chapman, M. D., Houston, Texas

4:15 P. M.

Panel Discussion: Hypertension, Systemic and Pulmonary

Drs. Chamberlain, Chapman, Deming and Waggenspack, Jr.

RUDOLPH MATAS AWARD IN VASCULAR SURGERY

A memorial service honoring Dr. Rudolph Matas and the seventh presentation of the Rudolph Matas Award in Vascular Surgery will be held Friday, May 9, in the Hutchinson Memorial Building of Tulane University School of Medicine. Dr. Matas, who died September 23, 1957, at the age of 97 years served Tulane's Department of Surgery all of his adult life, becoming Professor and Head of the Department in 1894. Upon his retirement in 1927 he was appointed Emeritus Professor of Surgery which post he occupied until his death. Dr. Matas was a member of the Louisiana State Medical Society for 77 years and its President in 1894 and was a member of the Orleans Parish Medical Society for 75 years. He was one of the twelve founders of the American College of Surgeons in 1913 and was its President in 1925-1926. He was President of the American Surgical Association in 1909, President of the International Society of Surgery in 1936, President of the American Association for Thoracic Surgery in 1920, and President of the Southern Surgical and Gynecological Association, now the Southern Surgical Association, in 1910. Among his honorary memberships were those in the Royal Academy of Medicine of Barcelona, the Royal College of Surgeons (England), the National Cuban Order of Carlos Findlay, the Order of Leopold of Belgium, the Gorgas Medical Society, the Havana Academy of Medicine, and the Spanish Royal Academy.

The Rudolph Matas Award in Vascular Surgery will be made to Dr. John H. Gibbon, Jr., the Samuel D. Gross Professor of Surgery and Head of the Department at Jefferson Medical College of Philadelphia. The award is in recognition of his pioneering efforts in the field of extracorporeal circulation which resulted in the development of a heart-lung apparatus. Recipients of the award in past years are Dr. Mont R. Reid, 1934; Dr. Reynaldo dos Santos, 1937; Dr. Daniel C. Elkin, 1940; Dr. Robert E. Gross, 1947; Dr. Alfred Blalock, 1950; and Dr. Michael E. DeBakey and Dr. Emile Holman, 1954.

THIRTEENTH NATIONAL CONFERENCE ON RURAL HEALTH

Heidelberg Hotel, Jackson, Mississippi

March 6-8, 1958

The Thirteenth annual conference was held in Jackson, Mississippi, March 6, 7, 8, 1958, in the Heidelberg Hotel. Due to the bad weather, and other conditions, the attendance was not quite as high as it had been in years gone by, but the enthusiasm was still there. The members of the Rural Health Council of the American Medical Association were there, and many of the Advisory Council members.

The first day's theme covered a nutritional balance for rural people, and a panel discussion later in the afternoon was given on "The Family Visits the Dentist". A reception was given that night by the Mississippi State Medical Society.

On Friday morning, the program consisted of two panels, one on "Safety" and the other on "What the Doctor Expects and What the Patient Expects of His Doctor". The afternoon was given over to the "Rising Costs of Health Service" and the "Distribution of Physicians". Dr. George E. Twente, of Jackson, Mississippi, went into the latter thoroughly, explaining the fact that there are plenty of doctors in America, but that for various reasons, many times economic, they do not locate in sparsely developed places. Since there is a greater production of doctors than there has ever been, he suggested that we might have a situation of surplus physicians.

The banquet on Friday night was presided over by Dr. F. S. Crockett, the perennial chairman of the Rural Health Council and Rural Health Conference. Two able speakers, Dr. Howard A. Nelson, President of the Mississippi State Medical Association, who spoke on "Medicine's Prestige" and Dr. F. J. L. Blasingame, General Manager of the American Medical Association, who spoke on "Patient-Physician Cooperation in Rural Health", were well received. The banquet was well attended, and the enthusiasm was great.

Saturday morning was given over to a number of subjects, including a paper by Mrs. John Rice of Sheldon, Illinois, 'We Work for Health". "Something Old; Something New" was presented by Mrs. Frances H. MacDonald of Oklahoma City. Dr. Paul M. Bancroft of Lincoln, Nebraska discussed "The Fourth "H" (Health). This was followed by a demonstration on the right way to handle injuries by a team of youngsters from Mississippi, and a preceptorship program as carried out by Cincinnati University.

The Conference was closed on a vibrant note by Mrs. Charles W. Sewell, the perennial speaker for the Conference and the "sweetheart of rural America", in which she discussed "It's Your Move".

The former chairman, Dr. Guy R. Jones of Lockport, Louisiana, and your author, Chairman of the Rural and Urban Health Committee, were in attendance. There were various other members of the Louisiana Health Council in attendance, representing the state of Louisiana.

J. P. SANDERS, M.D.

A.M.A. FILM REVIEWS

The ninth annual publication of "Reviews of Medical Motion Pictures" is now available on request from the Film Library of the A.M.A. This publication is prepared by the Council on Scientific Assembly, Motion Pictures and Medical Television, and contains reprints of all film reviews published in The Journal A.M.A. during 1957.

INSECTICIDE RESIDUE MAY CAUSE POISONING

More attention should be given to the possibility of poisoning from residue left on plants after they have been dusted with insecticides, two West Coast researchers said recently.

This was reported in an article written for the Feb. 15 Journal of the American Medical Association by Dr. Griffith E. Quinby, of Wenatchee, Wash., and Allen B. Lemmon, A.B., of Sacramento, Calif.

The warning of the danger from residues was advanced after the team studied a number of outbreaks over a six-year period of parathion poisoning among crop workers who entered fields shortly after the crops had been dusted with the chemical.

"Previously, due to the limited number of published reports of poisoning by residues, doctors have tended to insist on a history of direct exposure to sprays, concentrates, or dusts before giving serious consideration to a diagnosis of parathion poisoning," the researchers said.

A striking difference exists between poisoning produced by direct exposure and that due to residues.

In cases of direct exposure it is unusual for more than one or two of those involved to become ill.

Large numbers of those exposed to residues have been known to be affected. This type is also considered to be more mild and the onset is more gradual.

The current study also points out that of those crops examined, all had foliage at least chest high. "This may imply that workers are poisoned in this way only when dusted or bathed in the dilute residues practically from head to foot," the report added.

In the majority of the cases, poisoning was detected within two days after the solution had been applied to the crops. There have been extreme cases of poisoning up to four weeks after exposure.

Parathion is a chemical spray long used by workers who thin, pick, cultivate or irrigate crops. These crops include: apples, pears, grapes, hops, and citrus fruits.

The report concludes by saying, "It is obvious that regulations and recommendations which have been promulgated to prevent occurrence of such episodes are not only justified in purpose but probably need review, modification and improved enforcement if such incidents are to be prevented."

Dr. Quinby is attached to the Communicable Disease Center, Public Health Service, U. S. Department of Health, Education and Welfare, at Atlanta, Ga., and Mr. Lemmon is with the Bureau of Chemistry, Department of Agriculture of California.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

ORLEANS PARISH

The highlight on the agendum of entertainment for the wives of the visiting doctors in New Orleans attending the Twenty-first Annual meeting of The New Orleans Graduate Medical Assembly was the tea and fashion show.

The members of the Woman's Auxiliary to the Orleans Parish Medical Society entertained in honor of the visitors at a Mexican Fiesta Fashion show and Tea at the Orleans Club on Wednesday March 5th.

On the stage of the Auditorium of the Orleans Club the "south of the border" idea was carried out in a rainbow of color and glamorous cruise and resort ensembles with a sprinkling of spring and summer fashions, modeled to the delight of the visitors.

Receiving guests in the drawing room with the auxiliary president, Mrs. Eugene H. Countiss, were Mmes. Albert William Habeeb, Charles Lafayette Brown, Joseph O. Weilbacher, Jr., Charles B. Odom, Boni J. DeLaureal and H. Ashton Thomas.

Assisting in the dining room were Mmes. Martin O. Miller, Alton Ochsner, William Kohlmann, Mannie Mallowitz, James Ciaravella, Joseph J. Ciolino, C. Grenes Cole, J. Morgan Lyons, Esmond Fatter and Milton L. McCall.

Alternating at presiding at the tea and coffee services were Mmes. Max M. Hattaway, Andrew V. Friedrichs, Joseph D'Antoni, Edgar Hull, Edwin H. Lawson, Max Green, Maurice St. Martin, Ralph Hartwell, V. Medd Henington, H. Reichard Kahle, Donovan C. Browne, Val H. Fuchs, Charles Bahn, H. Theodore Simon and Jules Myron Davidson.

Mrs. Branch J. Aymond,
Publicity Chairman.

A cordial invitation is extended to all members of the Woman's Auxiliary to the American Medical Association, their guests, and the guests of physicians attending the convention of the American Medical Association, to participate in all social functions and attend the general meetings of the Auxiliary.

Headquarters will be at the Fairmont Hotel in San Francisco. The dates of the convention are June 23 - 27, 1958. Tickets for the various social functions will be available at the registration desk. There will be Hospitality rooms in the Green and Empire Rooms on the first floor.

Please register early and obtain your badge and program.

For further information contact:

(Mrs. M.) Harriet G. Mallowitz
State Chairman,
Press and Publicity

BOOK REVIEWS

Handbook of Physical Therapy; by Robert Shestack. New York, N. Y., Springer Publishing Co. Inc., 1956, pp. 119, $4.25.

This book is the outgrowth of the author's experience of fourteen years as a physical therapist. It is intended to present some of the fundamentals and uses of this rapidly growing science to the general practitioner, and also to be a quick reference for the physical therapist.

The book is divided into two parts. The first one describes the various modalities such as infra-red heat, hydrotherapy, massage, etc. available to the therapist. The second part takes up the application of these modalities to the treatment of common disorders. Completing the book is a section in which the muscles of the body are arranged according to their action.

The author is to be commended on his efforts to simplify the field of physical medicine for the busy physician and technician, but the result is over-simplification which may lead the reader to much misunderstanding. A chapter on the history of physical medicine is two pages long. No mention is made of the founding of the American Board of Physical Medicine—a milestone in the development of the specialty, or the creation of its own section in the A.M.A.

Chapter headings are particularly misleading. One chapter deals with "Neurological Conditions", another with "Peripheral Nerve Conditions"; yet brachial neuritis and the intervertebral disc syndrome are discussed under the former. Poliomyelitis, which certainly is a neurological condition, is separated from it by chapters on "Acute Joint Injuries and Orthopedic Disorders", and "Fractures". One is led to believe that fractures are not orthopedic disorders.

Oversimplification is carried to extremes in other instances. The chapter on the eye, ear, nose and throat contains this treatment for chronic meibomitis: "Massage of the lids is very helpful". The statement is true, but certainly does little to show the physician how to massage the lids, and how often.

Elsewhere, the author swings to the complicated tack. He states on page 117; "There are many tests for diagnosing nerve injuries, such as electromyography, chronaxie, dermometry, sign of Tinel,

reaction to galvanic and faradic current, and the sweating test. Special training and equipment are needed for some of these tests. The electrical and sweating tests can be easily performed in any physician's office". This reviewer knows of few tests more simple than Tinel's sign. It involves percussing over the site of a divided nerve. A tingling sensation distal to the percussion site indicates a partial lesion or the beginning regeneration of the nerve.

In summary, physical therapists and technicians will find the handbook very useful. Physicians would do well to use it only in conjunction with a standard textbook, like Krusen's "Physical Medicine", or Watkins' "Physical Medicine in General Practice".

JOSEPH P. RUMAGE, M. D.

The Early Diagnosis and Treatment of Acoustic Nerve Tumors; by J. Lawrence Pool, M.D., and Arthur A. Pava, M.D., Springfield, Ill., Charles C Thomas, 1957, pp. 161. Price $5.50.

In the historical section there is a reproduction of a plate showing a dissection of an acoustic neurinoma, an illustration from a book by Charles Bell published in 1833. Bell also called attention to an involvement of the fifth nerve in addition to involving the portio dura and the portio mollis of the seventh nerve, which were the original names of the seventh and eighth nerves. As this patient died from a respiratory condition, Bell classed the seventh nerve as a respiratory nerve. The library at the Tulane Medical School has a copy of the book published in 1833, as well as several others published by Bell. He found so many cases of facial nerve paralysis that the condition was aptly called Bell's palsy. He also described anesthesia of the cornea associated with involvement of the fifth nerve. In addition to the classification of portio dura and portio mollis used with the seventh nerve, originally they claimed four ossicles: (1) malleus, (2) incus, (3) orbicular, (4) stapes. In the "Structure, Economy and Diseases of the Ear" by George Pilcher, M.D., in 1843, the orbicular is represented by a straight line. In the "Outlines of Physiology" by J. L. Comstock, M.D., in 1836, the orbicular is pictured as a circle. Kirkes and Paget's "Physiology", published in 1853, dropped the orbicular. They also agree with Bell and say that the seventh nerve is not a nerve of respiration.

Isaac H. Jones, M.D., in his "Equilibrium and Vertigo", states that the majority of the tumors in the cerebellopontile angles are acoustic neurinomas.

While serving as Neuroto-ophthalmo-laryngologist, in Clarence A. Patten's Clinic at the Delaware Hospital, Wilmington, Delaware, 25 brain lesions were found. One of these was an acoustic neurinoma. The patient had the cardinal symptoms: ipsilateral deafness and an ipsilateral lack of response to the caloric test. In addition, was a marked ipsilateral papilledema with a decided increase of the size of the p .ysiological blind spot mapped out with a Peter's campimeter. This was confirmed when operated upon by Charles H. Frazier, M.D., at the University of Pennsylvania Hospital. Unfortunately, the patient died upon the sixth day. In discussing the case with Temple Fay, M.D., who was Frazier's resident, he sympathized with me when I told him that 2 out of 25 lived, by telling me that that was a good mortality.

In routine microscopic study of 250 petrous bones, Hady and Crowe report 6 in which extremely small neurinoma were present. (Hardy, M., and Crowe, S. J.: Early asymptomatic acoustic tumors, Arch. Surg. 32: 292, 1936). Lt. Colonel Theodore L. Hartridge, M.C., U.S.A., Brooke General Hospital, referred to the technique of Dr. Stacy R. Guild, Otological Dept., Johns Hopkins Hospital, in removing temporal bones for section. I am sure that his technique was used.

Fortunately these authors have made great strides in pathology, diagnosis, and decrease in operative mortality.

WILLIAM F. BONNER, M.D.

M.D., Eleventh Edition, The C. V. Mosby Com-*Diseases of the Skin;* by Richard L. Sutton, Jr., pany, St. Louis 3, Mo., 1956, 1479 pp. Price $29.50.

This voluminous textbook gives an excellent and modern account of dermatology. The subject matter is presented in a methodical and detailed manner, and a tremendous amount of material is included. There are 1972 good illustrations to supplement the reading material. These include both clinical photographs and photomicrographs.

One of the outstanding features of the textbook is its large and up-to-date bibliography which provides a convenient source of references when one wants detailed information on a subject.

This textbook is written primarily for those interested in advanced dermatology, but it would be useful as a reference book for physicians in general.

MARVIN E. CHERNOSKY, M.D.

PUBLICATIONS RECEIVED

Bailliere, Tindall and Cox, London, Eng.: Diagnosis and Treatment of Diseases in the Tropics, by H. C. Trowell, M. D. (3rd edit.).

Grune & Stratton, N. Y.: Theory and Problems of Child Development, by David P. Ausubel, M. D., Ph.D.; Vertigo and Dizziness, by Bernard J. Alpers, M. D.; Psychopathology of Communication, edited by Paul H. Hoch, M. D., and Joseph Zubin, Ph.D.

Philosophical Library, N. Y.: The Neuroses and Their Treatment, edited by Edward Podolsky, M. D.